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DYSENTERY: A PRACTICAL SURVEY OF ONE THOUSAND CASES IN A GENERAL HOSPITAL IN EGYPT, 1918-1919.[1]

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Historical Survey.

OUR real knowledge of dysentery is of comparatively recent date. Although Hippocrates was the first to describe accurately the disease which we now know as dysentery, and yet there is good ground for believing that the disease existed in Egypt and India for centuries before Christ. Many of the older writers failed to differentiate conditions which showed blood and mucus in the stools from those with blood alone. And it was only during the last century that authorities have considered the association of mucus with the blood as an essential in clinical diagnosis.

The ætiology and epidemiology of bacillary dysentery have been fairly definite since about 1900; amœbic dysentery, on the other hand, was not in such a secure position till about 1913. A brief historical note, therefore, may be of interest.

In 1752 Sir John Pringle published one of the earliest modern descriptions of dysentery, in his work "Observations on the Diseases of the Army", based on his experiences in Flanders. "The disease was worse in fixed camps at the end of the close, hot summer and in the autumn, when it was epidemic and contagious." He looked upon "heat and moisture of the air" as the remote causes, and a "putrefying state of the blood, and scurvy" as predisposing causes. He adds the following interesting remark:

> But having since perused the curious dissertation by Linnæus, in favour of Kercher's suggestion of contagion by animalculæ, it seems reasonable to suspend all hypothesis till that matter is further inquired into.

Over a century elapsed before this prophetic writing was fulfilled.

Annesley (1828) published his "Researches on the Diseases of India", containing a full description of dysentery and abscess of the liver as seen in Madras.

[1] Abridged from a thesis submitted to Edinburgh University for the Degree of Doctor of Medicine.

He found liver abscess *post mortem* in twenty-six out of fifty-one cases, and it is therefore probable that he was dealing chiefly with the amœbic variety of the disease. Sir Leonard Rogers says:

> Annesley's classical description is by far the best account of amœbic dysentery to be found in the older writings, if indeed it has ever been surpassed.

Twining (1835), Parkes (1846), and Mackinnon (1848) all described fully the disease in various parts of India, and Baly in his Goulstonian Lectures of 1847 gave a good description of dysentery in the Millbank Prison in London. The type he met with was clearly the bacillary form, and he considered that the outbreaks were "due to a local influence, malarial in nature, arising from the soil, and that it was not due to bad water, inadequate ventilation, or sewage defects".

Woodward (1880), in his "Medical and Surgical History of the War of the Rebellion", gives a very exhaustive account of diarrhœa and dysentery as it occurred in the United States of America.

Fayrer (1881), Maclean (1886), Chevers (1886), Kelsch and Kiener (1889), and numerous other writers have also given us excellent descriptions of the disease in various parts of the world. All these observers have shown that bacillary dysentery has always been the prevalent type of the disease in temperate climates, occurring especially in institutions and in military camps during times of war. In warm climates the prevalent type would appear to have been the amœbic variety, although bacillary cases were also met with.

It is hardly surprising that amœbic and bacillary dysentery were not differentiated from each other by these early workers, as the clinical symptoms of the two forms are so often indistinguishable. In 1859 Lambl first discovered "amœbic" forms of the organism in the human intestine, but controversy continued as to whether the organisms were the active causative agent in the production of the disease, or merely accidental or secondary concomitants.

In 1898 Shiga discovered the *Bacillus dysenteriæ*, and this, with other closely allied strains of the coli group, was proved to be the cause of the forms of dysentery which prevail in temperate climates as well as of a proportion of tropical cases. And not till about this time did the *Amœba dysenteriæ* come to be recognized generally as the agent in producing a large proportion of cases of dysentery in the tropics as well as in sub-tropical climates, and the discordant facts recorded by research workers in all parts of the world became reconcilable.

It may be of interest to recapitulate very briefly an outline of previous work on the treatment of dysentery. Sir John Pringle in 1774, when confronted with epidemic bacillary dysentery during the Walcheren Expedition, recommended moderate bleeding, careful purging, opium and ipecacuanha in hourly doses of 0·3 gramme (five grains).

Annesley (1828) also advocated bleeding, warm baths, calomel and opium. He appears only to have given ipecacuanha in 0·06 to 0·18 gramme (one to three grain) doses once or twice a day.

Other early workers, Twining (1835), Parkes (1848), Baly (1847), Maclean (1886), all gave full attention to the treatment of symptoms and had considerable success in the administration of ipecacuanha by the mouth.

Ipecacuanha, or Brazilian root, was first brought to Europe in 1658 by Piso and given in the form of an infusion or decoction. It appears to have been used in India as early as 1660, but later to have fallen out of use, both there and in Europe. Annesley and Twining in the early part of the nineteenth century employed it, as we have seen, and with significant success. But Docker is generally credited with having successfully reintroduced the drug in 1858, amongst British troops in Mauritius.

It is interesting to note that while ipecacuanha was so signally successful in certain cases, these observers noticed that many patients showed no improvement whatsoever. Now, of course, we know the cases to have been bacillary in origin. In such cases calomel and opium were the drugs most generally used. Maclean (1886) writes:

> How ipecacuanha, almost deserving the name of a specific, came to be superseded by calomel and opium in the treatment of dysentery in the East is one of the most curious questions in the history of tropical medicine.

The differentiation of the amœbic from the bacillary type of dysentery has furnished the solution to Maclean's problem.

Emetine, isolated by Pelletier in 1817, was used for the first time in the treatment of this disease by Bardsley (1829) and Tull-Welsh (1891), but its importance as a specific therapeutic agent in the treatment of amœbic dysentery was not fully recognized until Leonard Rogers reported its success in 1912.

At the time when the main part of this work was carried out, during the last two years of the Great War, ipecacuanha and emetine, administered orally and by injection, were both recognized as of undoubted value in amœbic dysentery. The bacillary forms were found to respond fairly well to specific therapy. During the past twelve years immense steps have been taken in the treatment of the dysenteries, but it yet remains to be seen whether in times of war our modern chemotherapy and bacteriophage treatment will enable us to combat even more successfully the "dreaded scourge of the camp".

Pathology.

Pathology of Amœbic Dysentery.

Amœbic dysentery is due to infection by the *Entamœba histolytica* and by it alone. No other amœba is capable of causing this disease, and it requires no help from secondarily infecting organisms of the bowel in the production of its lesions.

The life-history of this parasite is briefly as follows. The encysted form is swallowed with the food. Having arrived in the small intestine, the digestive juices there dissolve the capsule and set free the small daughter amœbæ, which make their way to the large intestine and thence down the lumina of the mucosal glands into the submucosa. There they grow into the adult or vegetative form, multiply by binary fission, and give rise to a direct colliquative necrosis

of the submucosa and *muscularis mucosæ* by means of a proteolytic ferment which they are able to secrete.

Direct communication with the lumen of the gut—that is, ulceration—is attained only when either the solvent attacks the intercellular cement of the mucosa or the necrosis extends far enough to interfere with its blood supply. According to which occurs first the mucosal cells are shed singly or *en bloc*, and an irregular ulcer with ragged, undermined edges is formed. The loss of substance is far greater in the submucosa than in the mucosa, as large vegetative amœbæ are found in the greatest numbers there; often sinuses lead from one ulcer to another under bridges of perfectly healthy mucosa.

As the amœbæ approach the surface of the mucous membrane again, they divide more and more rapidly, and consequently become smaller and smaller until on the surface of the gut they assume the "*minuta*" form. Many of them now encyst, and they are passed out of the body in the stools as cysts or *Entamœbæ minutæ*. It is worthy of note that the *Entamœbæ minutæ* produce no destruction of the tissues, and consequently no symptoms of dysentery; a fact which is distinctly in favour of the larger, or older, amœbæ producing a proteolytic ferment.

Adami was the first to point out that the reproductive and the higher functional activities of cells are mutually antagonistic; the more embryonic the cell the more readily does it multiply, which explains the more rapid growth of a neoplasm composed in greater part of cells of embryonic type. The smaller size of these amœbæ is due to their more rapid division, which has used up all their reserve of energy; consequently none is left for the development of that particular function which differentiates *Entamœba histolytica* from all other amœbæ.

It has never been shown that the entamœbæ produce a diffusible toxin, or that they are capable of exerting any poisonous effect other than the proteolysis of cells in their immediate neighbourhood. In consequence, the onset of the disease as a rule is insidious—it may be even unnoticed—and it is not until a comparatively large area of the gut is involved that the patient becomes at all acutely ill. It will thus be noted that the mucosal cells are in general healthy, apart from the areas of actual infections, while at autopsy the typical picture seen is one of areas of circumscribed necrosis, dotted like islands in the midst of healthy pink mucosa.

Since emetine treatment was universally adopted, cases where the bowel has undergone "gangrene *en masse*" are, fortunately so rare as to warrant only passing notice. This condition is brought about by an extensive amœbic invasion of the submucous and muscular coats of the intestine, with consequent thrombosis of blood vessels, and the blood supply of the bowel is cut off as effectually as by multiple thrombosis arising from any other cause.

The result is that the entire thickness of the bowel over a greater or lesser extent becomes gangrenous, and is passed as stringy black sloughs having a horribly fœtid odour. On very rare occasions it has even been impossible to remove the colon at autopsy, as all that remained to represent it was a slimy, stringy, black mass lying in a bath of corruption. Unless one had actually seen such a case it would be inconceivable that a man could survive long enough for his intestines and his peritoneum to attain such a state.

It has frequently been shown that fatal cases of amœbic dysentery often terminate with a blood infection by members of the *Streptococcus fæcalis* group. There is nothing astonishing in this if one considers how often the intestinal bacteria invade the body before death in different chronic and subacute diseases. The astonishing part is that it does not occur more frequently in amœbic dysentery—a disease in which there is such an extensive solution of continuity of the mucosal barrier. The point which the writer would like to emphasize is that in the vast majority of cases of non-fatal amœbic dysentery no such invasion of the blood stream can be demonstrated by blood culture.

This may be due to one or two causes acting together or separately. In the first place, the liver may act as an efficient bacterial filter; in the second, the bowel itself is probably endowed, even when diseased, with a high immunity against just such organisms. Wright has shown that different parts of the body possess different degrees of resisting power against pyogenic organisms. It seems reasonable to suppose that this local immunity acquired by countless minute inoculations with the bacteria-containing fluid in which the mucosa has been bathed since birth, is sufficient to protect not only the body against invasion by these bacteria, but even the mucosa itself when the latter is partially destroyed by amœbæ.

In amœbic dysentery only when extensive areas are killed by interference with their blood supply, does any secondary infection of the mucosa take place, just as a limb may become affected by wet gangrene after the blocking of its blood supply. When, however, a pathogenic organism, such as a dysentery bacillus, which is a stranger to the mucosal barrier, and against which the mucosa is in consequence not immunized, arrives on the scene in sufficient numbers, virulence with its destructive properties immediately comes into play.

When sections are made through the edges of early amœbic ulcers, and more particularly when the unbroken amœbic nests in submucosa or liver are examined histologically, the observer is struck by the absence of the usual cellular infiltration characteristic of inflammatory reaction. This is all the more noticeable when a comparison is made with the corresponding sections of early bacillary dysentery which is an inflammatory disease.

Again, the so-called amœbic liver "abscess" when sterile is not an abscess at all. It is a primary colliquative necrosis of hepatic tissue, which is broken down and changed by a proteolytic enzyme from protein to albumoses and peptones; the patient's symptoms are due to the absorption of these poisons directly into the blood stream and can be reproduced experimentally by the intravenous injection of peptone.

To sum up: the pathology of histolytica amœbiasis is a primary degeneration due to the chemical digestion

of the cells in the immediate neighbourhood only of the amœbic enzyme. It is not a primary inflammation with consequent degeneration; any inflammation which occurs is due to secondary microbic infection, is generally not severe, and takes place late in the disease.

The nature of the cellular exudate in the stools and in the uncontaminated liver "pus" is in entire accord with this basal fact of its pathology.

Pathology of Bacillary Dysentery.

Very different from the amœbic lesions are the changes which occur in bacillary dysentery. The infecting agents belong to a different kingdom.

The two diseases are so totally distinct that the only excuse for grouping them both under the heading of "Dysentery" is that of historical usage. In logic it were as reasonable to group cancer and tuberculosis of the intestine under the same heading because they may both give rise to what used to be called the "bloody flux".

Whatever may be the route of infection the essence of the pathology of bacillary dysentery is the intense diffuse catarrhal inflammation of the mucosa, which according to the severity of the attack may resolve or may go on to degenerative changes, such as coagulation necrosis and sloughing. It is a primary inflammation of bacterial toxic origin, with all the cardinal signs of this condition, namely, "*rubor, tumor, calor, dolor et functio læsa*".

The process is essentially one of diffuse destruction of the superficial layers of the mucosa, accompanied by œdema, large immigration of leucocytes, diapedesis of blood cells, and hæmorrhagic extravasation—such as is seen in all cases of acute bacterial inflammation of tissue. The immigration of polymorphonuclear cells is an especially noteworthy feature. At autopsy the appearance of the intestine in any case of bacillary dysentery will vary with the intensity of the infection and the length of time the patient has survived it.

In the so-called fulminating ileo-colic dysentery, the entire surface of the lower part of the ileum and large intestine, excepting Peyer's patches and the appendix, is seen to be the site of intense inflammatory change. No part escapes, as it does in amœbic dysentery; the bowel wall is œdematous, enormously thickened, deep plum-red in colour, and the mucosa may resemble the surface of a large granulating wound. A case of this intensity is more likely to be met with in patients who happen to be affected with renal disease of long standing, and death takes place from poisoning before the changes later to be described have had time to occur.

If the patient survives this stage, the superficial layers of the entire mucosa undergo coagulation necrosis, become bile-stained, and are replaced by a green diphtheritic false membrane which may entirely hide the red granular layer underneath it. Here or there smaller or larger areas of this membrane may have sloughed, disclosing buds of granulation tissue of greater or lesser extent. Coincidently, small but numerous intramucosal hæmorrhagic extravasations take place.

If the patient again survives this stage, small superficial ulcers, affecting particularly the summits of the *valvulæ conniventes* and mucosal folds, are formed. The minutely eroded, pinkish-grey "coraline" appearance of these ulcers, which run transversely across the bowel along the ridges of the mucosal folds of the colon, and are often present in large numbers, confers a characteristic appearance. The sloughed mucosa is gradually replaced by well-formed and vascularized granulation tissue, and later by a fibrous hyperplasia. If the patient recovers entirely, the large intestine becomes shrunken into a thick-walled firm tube, with narrowing of its lumen and permanent damage to its functions.

Again, the ulcerative process may continue and spread, with the result that the gut wall may be honeycombed with myriads of small ulcers and so finally resemble a piece of red porous rubber. These ulcers, in spite of the fact that they may give rise to the so-called "worm-eaten condition", can easily be distinguished from the amœbic ulcers by the fact that they are more superficial, more regularly circular or oval, and have cleaner cut edges than the ragged undermined amœbic ulcers. Their depth will vary with the amount of fibrous change that has taken place in the bowel wall around them, which may be so great as to make them appear deeper than they really are. Whereas the thickened peritoneum may often constitute the base of a healed amœbic ulcer, it is extremely rare to find a bacillary ulcer penetrating deeper than the *muscularis mucosæ*. Perforation or peritonitis is practically unknown in pure bacillary dysentery.

Varying with the intensity of the infection, certain areas of the intestine are affected more than others. Rogers states that the descending colon and rectum are more frequently involved than any other part. In my experience, on the other hand, the caecum, first part of the ascending colon, and the flexures have been more profoundly affected than the other parts of the large gut.

When complete recovery has occurred after a less extensive infection than that first described, the mucosa is often seen to be atrophied and stained a dirty slate-grey colour as a result of the diffuse intramucosal hæmorrhagic extravasation. In healed amœbic ulcers, on the other hand, this staining is seen only in and immediately around the cicatrices of the ulcers.

Concurrently with the local changes are those produced in heart, liver, kidneys, adrenals, and probably the nervous system, due to the absorption of the toxins formed in the gut by the dysentery bacilli.

In bacillary dysentery, as in diphtheria, the patient dies of intoxication. It may be definitely stated that as a general rule dysentery bacilli do not invade the blood stream, except during the stage of premonitory fever before diarrhœa begins. On one or two occasions certain observers have claimed to have isolated them by blood culture; but this invasion of the blood must be looked on as an accident in the course of the disease, and the occasions on which a general dysenteric bacillæmia takes place are so rare that the probability of its occurrence is negligible.

Just as the amœba is responsible for the ulcers of amœbic dysentery, so is the dysentery bacillus

accountable for the damage to gut and general system. I do not deny that secondary infection of the blood stream may and does take place occasionally in the course of both diseases, but we controvert the idea that any such infection is to be regarded as a regular concomitant.

It has been known for many years—in fact, since Todd first worked out the poison of Shiga's bacillus—that the non-mannite-fermenting type of dysentery bacillus produces a soluble toxin. Whether this toxin is really extracellular and diffusible from the first or whether it becomes so owing to a rapid breaking down of the bacillary bodies and consequent liberation of endotoxin, is a matter of no moment. The fact remains that a filtrable toxin is easily demonstrated in cultures of this bacillus.

Everyone who has had experience of dysentery due to the mannite-fermenting type of bacilli must have been forced to the conclusion that a similar and equally virulent toxin was at work in these cases, as in those in which Shiga's bacillus was the morbific agent. The clinical symptoms are the same, and the degenerative changes in the organs seen at autopsy are indistinguishable from those seen in cases of death from Shiga's bacillus.

Diagnosis.

It will thus be seen that it is easy to differentiate the two great types of dysentery when they reach the *post mortem* table, so definite and peculiar to each is its morbid anatomy. It was hoped, therefore, that on *a priori* grounds the differences in the nature of the exudate and desquamated cells found in the stools of each variety would be sufficiently constant and characteristic to constitute a basis for a more rapid differential diagnosis, and investigations both in my laboratory and in others throughout Egypt have led me to the conclusion that it is possible to arrive at such a diagnosis, based on the cytological picture peculiar to each type of the disease.

The character of any given stool will depend upon the particular stage of the disease at which that stool has been evacuated. It will depend to a much less extent upon the nature of the food which the patient has taken, in that in the vast majority of cases both types of patients—those with amœbic and those with bacillary dysentery—will have been treated on similar dietetic lines; further, our examination is restricted to any sloughs, bloody mucus *et cetera* which may be mixed with the fæcal matter.

Cyto-Diagnosis in Amœbic Dysentery.

If some of the blood-stained mucus from a case of "simple" amœbic dysentery be examined microscopically, the first thing noticeable will be the scantiness of the cellular exudate, although red blood corpuscles may be very abundant. This point has particularly impressed me when examining material taken from the surface of an ulcer at autopsy.

Another salient characteristic is the preponderance of mononuclear cells over polymorphonuclear cells, a feature which contrasts sharply with the picture seen in bacillary dysentery.

Next to the scantiness of the cellular exudate the most noteworthy element in the film is the picture presented by the actual cell components—most of them look half-digested, and have a "mouse-eaten" appearance. The process evidently begins at the periphery, and the nucleus is the last to be involved. In many cases groups of nuclei appear to become agglutinated by means of their semi-digested surrounding cytoplasm. The process is apparently a slow one, the cytoplasm being gradually digested and dissolved and the unchanged nucleus being left bare or clothed merely in a narrow strip of ragged cytoplasm.

As will be shown later, the cells in a bacillary stool give evidence of being poisoned *en bloc* and undergo rapidly the changes characteristic of toxic necrosis and autolysis, the nucleus being among the first parts of the cell to be affected.

Further, the fatty changes to be described as occurring invariably in cells composing the bacillary exudate are never seen to anything like the same extent in amœbic stools when examined fresh in Sudan III solution; all that one sees are isolated "lumps" or dots of fat, suggesting that the cells had previously ingested some fatty material rather than that it was of intrinsic cytoplastic origin.

Consequently, all the evidence goes to prove that every morbid change seen in amœbic dysentery can be explained on the hypothesis of proteolytic digestion of living tissue by the amœbæ.

The characteristics of a "simple" amœbic stool may be summarized as follows:

1. Scantiness of cellular exudate, especially the polymorphonuclear element.
2. Preponderance of mononuclear over polymorphonuclear cells.
3. Evidence of proteolytic digestion of the cells, beginning at the periphery and affecting the nucleus last.
4. Absence of all phenomena characteristic of inflammatory reaction, toxic necrosis and consequent autolysis.

Cyto-Diagnosis in Bacillary Dysentery.

A very different picture is seen on examining microscopically the blood-stained muco-pus from a case of bacillary dysentery, whether in the acute, subacute, or chronic stage.

Here the first feature to strike the observer is the abundance of cellular exudate, composed largely of polymorphonuclear leucocytes. Desquamated and degenerated mucosal cells are also common, but the polymorphonuclear neutrophiles predominate. Blood may or may not be present according to the stage of the disease, whereas it is hardly ever absent from the stools of amœbic dysentery.

The polymorphonuclear neutrophiles show the characteristic changes consequent on toxic necrosis more than any other leucocytes, the eosinophile cells show less change and the lymphocytes least of all. The cell is evidently poisoned *en masse*, and whether the further changes about to be described are directly caused by the toxin or are merely autolytic phenomena resulting from the death of the cell is of no moment. The important point for our purpose is that the process is obviously different from that which takes place in the cells of an amœbic exudate.

The nuclei seem to be specially sensitive to the action of this toxin, as the changes described, particularly fragmentation and agglutination of small chromatin masses round the nuclear membrane, and "ringing" of the nuclei, have not been observed in similarly prepared films of pus obtained from other sources, for example, boils, or at least to nothing like the same degree.

A type of cell which is always present, often in large numbers, and which seems to be associated more frequently with bacillary than with amœbic dysentery, somewhat resembles a free resting amœba, and it is important not to mistake it for one.

There is, in my opinion, little doubt that this mistake has been often made, and that as a result the numbers returned as amœbic dysentery in 1915 were unwarrantably high. Particularly do I consider these remarks applicable to cases of so-called running "amœbic dysentery", often accompanied by a rise of temperature, which responded beneficially to serum treatment. The cells may have also given rise to the diagnosis "post-amœbic colitis".

As the cells are larger than a polymorphonuclear leucocyte, and as the changes in them are much the same as those occurring in other leucocytes, it will be convenient to describe them as a type.

They are very large mononuclear cells, 15 to 30μ in diameter, with basophilic non-granular cytoplasm, and are probably macrophages of endothelial origin.

The cytoplasm, if not too degenerated, contains numerous vacuoles in which there may be entire polymorphonuclear leucocytes, lymphocytes, red cells, or their fragments, and bacteria, more or less digested. Curiously enough, the ingested leucocytes are much less degenerated than their hosts. By Achard's method of eosin staining these large cells can usually be demonstrated to be dead. In others, as a result presumably of more advanced degeneration, the cytoplasm, instead of being granular and vacuolated, is swollen, hyaline, transparent, and apparently in a state of hydropic degeneration; it stains very poorly or not at all with iron alum hæmatoxylin.

The degenerative process begins early in the cytoplasm, which first becomes granular, then vacuolated, and finally hyaline. It may show dots of granules, which are greenish when examined in the fresh state and intensely black when stained with iron hæmatoxylin.

These are probably fragments extruded from the pyknotic nuclei. In fresh specimens these dots are frequently seen to exhibit distinct Brownian movements and are apt to be mistaken for bacteria or other *débris* which the cell has ingested. Coincidently with the cytoplasm the nuclei begin to show very definite karyorrhectic and karyolytic changes, both in fresh and stained specimens.

The nucleus first becomes swollen, then granular; finally its centre becomes transparent and the chromatin is aggregated under the nuclear membrane, leaving the centre bare. The nucleus then bursts, and the granules resulting from this action appear as a scattered line of irregular dots—green in the fresh and black in the stained specimens, which are apparently caught in the periplast, all that remains of the cytoplasm.

Later these dots disappear, and the cell is represented only by the periplast, which is usually regularly circular. These cells, as their suggested origin implies, possess certain feeble powers of amœboid movement, a characteristic which is liable to entrap the unwary.

These endothelial macrophages resemble resting or dead amœbæ most closely when they show the aggregation of chromatin granules scattered around the periphery of the nucleus; then one sees a large round cell, vacuolated and containing ingested cells, with a nucleus which exhibits the "ringing" so characteristic of the nucleus of amœbæ. The fact, however, that the nucleus, in a degenerating amœba, is the last structure to disappear may be of some guidance in differentiation.

Similar degenerative changes to those mentioned occur in the leucocytes and also to a less extent in the mucosal cells.

Fatty degeneration is always present in the cells composing the exudate in bacillary dysentery. In the very early stages this change is very slight and the intracorpuscular globules present in leucocytes and desquamated mucosal cells appear as dust-like red granules when examined under the immersion lens, when Sudan III in alcoholic solution is run in under the coverslip.

Later these globules become larger and larger, until in the advanced cases the greater part of the cell body is filled with them. In some I have even seen the leucocytes distended with large red globules crammed together inside the periplast like potatoes in a sack. This condition is best seen in the liquid green pea soup type of stool, without obvious mucopus or blood, characteristic of advanced subacute or chronic bacillary dysentery.

Another point to strike the observer in cases of acute bacillary dysentery is the extraordinarily small number of bacteria present; the film, in fact, looks more as though it had been taken from the pus of a streptococcal abscess than from a stool.

The characteristics of a bacillary stool may be summarized as follows:

1. Abundance of cellular exudate, mostly polymorphonuclear.

2. Preponderance of polymorphonuclear over mononuclear cells.

3. Evidence of toxic necrosis of cells, the degenerative changes occurring early in all parts of the cell, including the nucleus. This degeneration may go so far as to leave only the circular periplast of the cytoplasm, thus constituting the "ghost cell", the presence of which in any quantity is very typical of bacillary infection.

4. Evidence of phenomena characteristic of intense inflammatory reaction to microbic infection.

Treatment.

Treatment of Amœbic Dysentery by Emetine Bismuth Iodide.

The treatment of carriers of *Entamœba histolytica* with the double iodide of emetine and bismuth was originally tried at certain convalescent hospitals in England early in 1918, and at the time when this work was carried out was more or less on its trial. It was

of interest, therefore, to try to estimate its value as a curative agent in some of our cases in Egypt.

It has been shown that emetine hydrochloride, administered hypodermically even in full doses, was successful in about one-third only of the cases treated, and the following investigations, while of little more than historical interest now, at that time were of considerable significance.

To appreciate the results of treatment with this drug it must be noted that this series of patients was given emetine bismuth iodide because they had proved refractory to ordinary emetine injections, being still infected after receiving large doses of emetine hydrochloride hypodermically.

It was not understood at that time that the drug should be used in large quantities if the treatment was to be in any measure successful. Of emetine hydrochloride 0·06 gramme (one grain) is approximately equal, in emetine content, to 0·18 gramme (three grains) of the double iodide, and consequently, to obtain the equivalent of a full course of twelve grains of emetine injection, some thirty-six grains of the double iodide had to be administered to the patient.

Table I shows a series of ten cases which received 1·4 grammes (twenty-four grains) or less of the double iodide—the equivalent of approximately 0·52 gramme (eight and three-quarter grains) or less of the hydrochloride.

Table I.

Case Number.	Total Amount of Drug Given.	Result.
1	0·84 gramme (14 grains)	No effect.
2	0·84 gramme (14 grains)	Relapse after fourth day.
3	0·48 gramme (8 grains)	No effect.
4	0·84 gramme (14 grains)	No effect.
5	0·72 gramme (12 grains)	Relapse after fifth day.
6	0·48 gramme (8 grains)	No effect.
7	1·44 grammes (24 grains)	Relapse after eighth day.
8	0·36 gramme (6 grains)	No effect.
9	0·96 gramme (16 grains)	Relapse.
10	0·72 gramme (12 grains)	Relapse.

It will be seen that there is not a single successful treatment in the whole series; either there was no effect, or a relapse occurred in every case, and the conclusion to be drawn from these facts is obvious.

Table II shows the results of treatment carried out in twenty-four cases. Each patient received altogether a minimum amount of 1·92 grammes (thirty-two grains).

From this table it may easily be seen that the results were much more satisfactory than in the previous series of cases. It seemed reasonable to conclude, therefore, that emetine bismuth iodide, given in sufficient quantities, was considerably successful in the treatment of carriers of amœbic dysentery.

From the small number of cases and from the varied results of other observers it was too soon to claim that this drug was a certain means of cure. But there is no doubt that it was much superior to the older treatment with emetine injections, as most of these patients had previously been given the hydrochloride, often for a prolonged period, without success.

Only two patients with acute amœbic dysentery were treated with the double iodide. One of these had previously had a full course of emetine injections (0·06 gramme or one grain a day for twelve days). Both patients were rapidly and satisfactorily cured. The results were promising, but unfortunately no further cases were available for trial during my time in Tel-el-Kebir.

Table II.

Results of Treatment with Emetine Bismuth Iodide in Larger Quantities.

Case Number.	Total Amount of Drug Given.	Result.
1	2·16 grammes (36 grains)	Cured
2	2·16 grammes (36 grains)	Cured
3	1·92 grammes (32 grains)	Cured
4	2·52 grammes (42 grains)	? Cured
5	2·40 grammes (40 grains)	Not cured
6	1·92 grammes (32 grains)	? Cured
7	2·40 grammes (40 grains)	Cured
8	2·40 grammes (40 grains)	Cured
9	4·08 grammes (68 grains)	Cured
10	2·16 grammes (36 grains)	Cured
11	2·52 grammes (42 grains)	Cured
12	2·16 grammes (36 grains)	Cured
13	2·16 grammes (36 grains)	? Cured
14	2·40 grammes (40 grains)	Cured
15	1·92 grammes (32 grains)	? Cured
16	1·92 grammes (32 grains)	? Cured
17	2·16 grammes (36 grains)	Cured
18	2·16 grammes (36 grains)	Cured
19	2·16 grammes (36 grains)	? Not cured
20	2·40 grammes (40 grains)	? Cured
21	1·92 grammes (32 grains)	? Cured
22	2·16 grammes (36 grains)	Cured
23	2·16 grammes (36 grains)	Cured
24	1·92 grammes (32 grains)	Cured

We may therefore conclude:

(1) Emetine bismuth iodide, properly administered, has successfully cured the majority of carriers of *Entamœba histolytica* who have hitherto been treated.

(2) For treatment to be successful, the drug must be given in large quantities—not less than 2·16 to 2·40 grammes (36 to 40 grains), in daily doses of 0·18 to 0·24 gramme (three to four grains). Less than this amount is seldom efficacious, and more may be necessary in individual cases.

Treatment of other Protozoal Infections by Emetine Bismuth Iodide.

There is little to say concerning the treatment of any protozoal infection other than that with *Entamœba histolytica*. It seems highly probable that no method has yet been devised for removing any other protozoal inhabitant of the human bowel. There are, however, certain treatments which have been tried and found wanting, and which are therefore worthy of mention as they can now be definitely discarded as useless. Some of these may be briefly noted.

Entamœba coli.—It is clear from all the records in which cases have been followed for any length of time that treatment with emetine hydrochloride hypodermically will not remove *Entamœba coli*. When due regard is paid to the frequency with which negative examinations may be made on untreated persons infected with this organism, it appears highly probable that hypodermic injections of emetine have no effect whatever upon it. Cases may, indeed, become "negative" during and after treatment; but sooner or later they become positive again, and if daily examinations have been made during both treated and untreated periods there appears to be no significant correlation between "negative" examinations and treated periods.

There is, however, some evidence that emetine bismuth iodide occasionally removes *Entamœba coli*. Several cases have been recorded in which treatment with this drug has been followed by the complete disappearance of both *Entamœba histolytica* and *Entamœba coli* from the stools.

Of the patients who were treated with emetine bismuth iodide for *Entamœba histolytica* infection, thirteen were infected with *Entamœba coli*. Of these, three were not followed for a sufficient time to determine the possible effects of the drug upon their *Entamœba coli* infections. The other ten patients behaved as follows: one was positive every time he was examined, both during and after treatment; eight became "negative" during treatment and for a variable period afterwards, and then became positive again; one became "negative" during treatment and has remained "negative" ever since.

The remarkable coincidence between the "negative" period and the time of treatment in eight of the cases, and the apparently complete disappearance after treatment in one case, lead one to suppose that emetine bismuth iodide is not without effect upon *Entamœba coli*. The fact that nine patients out of ten treated were, however, certainly not freed from infection, leads one also to suppose that the effect is rarely permanent.

I find no evidence, based on adequate numbers of negative examinations, that any other method of treatment has any effect upon *Entamœba coli*.

Lamblia.—There is a considerable amount of evidence to show that no method of treatment has yet been found that will remove a lamblia infection. It is well known, when dealing with "negative" results of examinations that a very large number of examinations, must be made before it can be said with certainty that anybody is not infected with this organism. Examinations in untreated cases so frequently fail to reveal an infection that it is necessary to examine an infected person for a very long time to demonstrate that any treatment which he may undergo has any effect whatsoever upon his infection. A few "negative" examinations are without significance.

Many of the infected men under my observation have been treated with emetine hypodermically for *Entamœba histolytica* infection. There is abundant evidence to show that this treatment is without effect on lamblia. As there is not even one possible "cure" in any cases where numerous examinations have been made, it will be unnecessary to enter into details. Emetine bismuth iodide seems likewise without effect upon lamblia. It thus seems clear that emetine compounds have no effect whatever upon lamblia.

Trichomonas and Chilomastix.—There is no infection with trichomonas or chilomastix, in any series, which appears to have been cured by any treatment employed. Emetine, either hypodermically or as the double iodide, has no apparent effect upon either. This seems worthy of note. But as no claims to success have been made for any treatment, and as no reports deal specifically with the treatment of these infections, it will not be necessary to consider this subject here in greater detail.

We may conclude:

(1) Emetine bismuth iodide may exceptionally prove successful in removing an infection with *Entamœba coli*. As a rule it is not efficacious. No other drug has yet been shown to have any action upon this organism.

(2) There is no evidence, from the cases in the present series, to show that a successful method of removing flagellate infections (lamblia, trichomonas chilomastix) has yet been found.

The Serum Treatment of Bacillary Dysentery.

It is not necessary in this paper to discuss in detail the general treatment of acute bacillary dysentery, but my observations on the results of serum therapy may be of interest.

It was my custom to give heroic doses of serum to all new patients admitted to my wards with acute diarrhœa and blood and mucus in the stools. I did not wait for detailed pathological reports apart from an immediate stool examination, which, as has been already seen, was of the greatest value in clinching the diagnosis.

Sixty to eighty cubic centimetres of anti-dysenteric serum, supplied to us from the Lister Institute and prepared from the Shiga bacillus and one or more strains of Flexner bacillus, followed by one hundred and fifty to three hundred cubic centimetres of normal saline solution, were given twice daily for the first two days and once daily for the two following days.

There is no doubt (this was written some ten years ago) that this giving of serum was in some cases merely a ritual, as we were in the dark about the exact typing of the different strains of organism which were casual in our part of the country. But I do maintain that the life of a patient suffering from acute dysentery depended very often on the giving of serum at the earliest possible moment.

It is a practical impossibility to examine bacteriologically all cases of dysentery occurring among troops on active service, especially when they are scattered over wide tracts of country, and, further, a "negative" result will most probably ensue if the proper precautions are not adopted for a correct examination of the specimen.

Under the most favourable conditions a competent bacteriologist can determine in twelve to sixteen hours whether an infection is due to the Shiga or Flexner bacillus. This fact, however, is of little value under the conditions which exist with an army in the field. If, however, every patient with acute dysentery who required serum received a mixed antiserum to begin with, and all those with proved Shiga infections had received anti-Shiga serum, better results might have been obtained.

During the Gallipoli campaign, and in Macedonia during 1916, 1917 and 1918, the vast majority of cases of bacillary dysentery were caused by the Shiga bacillus. In our hospital this was also our experience.

Among approximately two hundred consecutive patients with bacillary dysentery who received serum our death rate was 1·5%. In the vast majority of instances the patients did not arrive at the hospital

until after the third day of the disease, some patients often having twenty or more blood and mucus stools per day. These figures, therefore, are of real significance.

In the cases that responded best, the temperature fell within twelve hours of the initial dose. The toxæmic symptoms, such as collapse and prostration, were satisfactorily alleviated. There is no doubt that the intravenous administration of saline solution was a factor of great significance in the improvement which was noticed. The distressing abdominal symptoms such as pain, tenesmus and urgent call to stool, all appeared to be more rapidly removed than in our previous experience, when we had no serum available.

There is no doubt, therefore, that the early neutralization of toxins in patients suffering from severe bacillary dysentery by the giving of a potent anti-dysenteric serum is an essential factor in the treatment of the disease.

Chlorine Water as an Adjunct in the Treatment of Dysentery.

The use of chlorine water lavage was advocated many years ago by Burney Yeo in the treatment of typhoid fever, with satisfactory results. This idea was applied in the treatment of infantile diarrhœa, and in a paper on the subject which I published in *The British Medical Journal* (December 15, 1917), it was pointed out that a large percentage of cases responded well to chlorine lavage, and it occurred to me therefore that a similar treatment, used of course in combination with emetine and serum, might be of value in dysentery. The following results, which were embodied in a report to the Senior Medical Officer in the Division, may therefore be of interest.

In this part of my investigation notes were made on five hundred and two patients with dysentery, of varying severity, who received this form of treatment, in addition to the routine measures.[1] Of these four hundred and four were bacillary infections, eighty-eight were amœbic, and ten were both bacillary and amœbic.

Seventeen patients died, thirteen with bacillary and four with amœbic and two with both bacillary and amœbic infections. Of the thirteen bacillary cases in which death occurred, three were complicated with pneumonia, two with tuberculosis, and three with pellagra. Of the four cases in which death occurred from amœbic dysentery, three were complicated with pulmonary disease and one with malaria.

At the time of this special investigation there was an abnormal number of daily admissions, and most of the patients being admitted in a very severe and advanced stage, the death rate was accordingly higher than would have been the case if the patients had been treated at an earlier stage of the disease.

In addition to the dysentery cases one hundred and six cases of acute diarrhœa came under observation; in fourteen of these lamblia cysts were reported. These cases also responded to chlorine water lavage in a highly satisfactory way.

Of the four hundred and four patients with bacillary dysentery in this series, two hundred and eighty-six were treated with both chlorine water lavage and antiserum, fifty with chlorine water lavage only, and sixty-eight with antiserum only. The cases were of more or less equal severity. There is absolutely no doubt that the patients who received both serum and chlorine water lavage were benefited to a much greater degree than either those who received serum or lavage only. (See charts, which are representative of all this series of cases.)

In a series of eighty-eight cases of amœbic dysentery of approximately equal severity, forty patients received chlorine lavage in addition to emetine or emetine bismuth iodide, and the remainder received only emetine. As in the previous group of cases, the results were highly satisfactory. Those who received chlorine water lavage were certainly more rapidly improved with earlier alleviation of symptoms.

Post Mortem Evidence.—Autopsies were performed in a number of cases. The results here were most striking. In the cases in which chlorine water lavage had been used the rectal and intestinal walls showed ulcerated areas which were definitely in the process of healing, evidenced by the presence of well-formed vascularized granulation tissue, and scars of recently-healed ulcers. In those cases in which no rectal chlorine water was used the ulcers presented by no means such a healthy picture. The ulcerated areas were well defined, superficial, with basal patches of necrosis. The surrounding tissues showed sloughing, along with marked hyperæmia and intense intramucosal hæmorrhagic extravasations. There is no doubt that, even in fatal cases, the local condition in the intestinal wall is very much improved by the use of chlorine water lavage in this condition.

Lavage with other Intestinal Antiseptics.—Lavage with normal saline and potassium permanganate (1 : 1000, 1 : 2000, 1 : 3000 solutions) was also tried in several patients, but with no success. The clinical condition was unimproved.

Summary and Conclusions.

1. A brief account is given of the historical aspect of dysentery, with regard to the clinical picture, diagnosis and treatment of the disease.

2. The comparative pathology of amœbic and bacillary dysentery is based on the author's personal examination of over twenty cadavera and several hundreds of stools. The value of cytology in diagnosis is discussed, and the conclusion is arrived at that differentiation between the two great types of dysentery is possible by simple stool examination. It is not claimed that this is a final means of proof, but that, during the stress of war conditions, it should be employed as an adjunct in the determination of subsequent treatment.

3. The uses of emetine bismuth iodide in the treatment of carriers and chronic cases of amœbic dysentery are discussed, and from the author's results this drug may be held to be of proved value in therapy. Serum in the treatment of acute bacillary dysentery is of undoubted value, as was seen from the low death rate in a series of two hundred acute cases. Chlorine water lavage as an accessory in the symptomatic treatment of acute dysentery has been

[1] In lavage a 0·5% solution was used two to four times a day.

SERUM

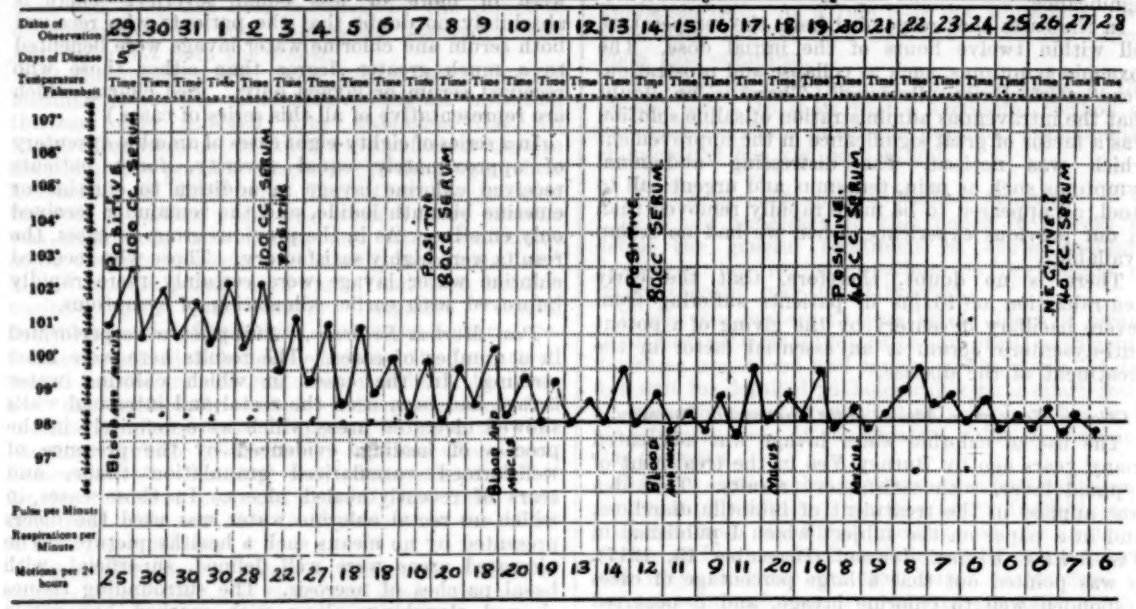
CLINICAL CHART.
(To be attached to Case Sheet.)

Army Form B. 181.

Corps _____ Military Hospital 7th P.O.W.

No. 7104 Rank and Name PTE K. E Age 27 Service 3 YEARS

Disease BACILLARY DYS. Date of admission DEC. 29 1918 Date of discharge _____ Result _____



Signature _____ In charge of case.

CHART I.

EMETINE.

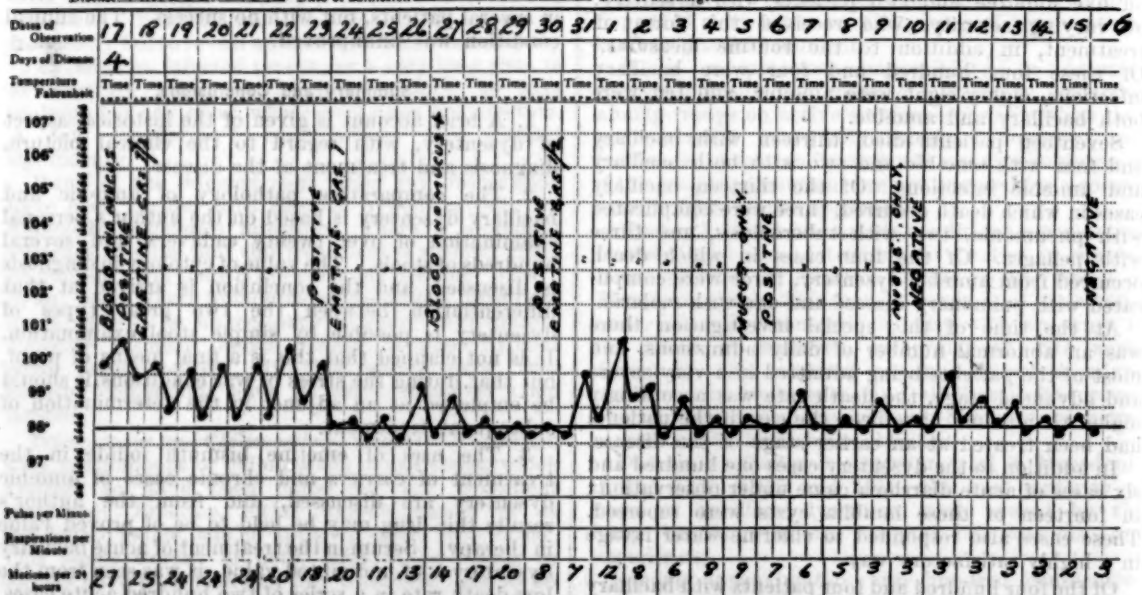
CLINICAL CHART.
(To be attached to Case Sheet.)

Army Form B. 181.

Corps _____ Military Hospital No 7 P.O.W.

No. 332 Rank and Name C.Pl. M. F. Age 25 Service 4 YEARS

Disease AMOEBIC DYS Date of admission 17th 12th 18 Date of discharge _____ Result _____



Signature _____ In charge of case.

CHART II.

CHLORINE AND SERUM

CLINICAL CHART.
(To be attached to Case Sheet.)

Army Form B 181.

Corps _____ Military Hospital P. O. W. No. 7

No. 6998 Rank and Name PTE H. R Age 24 Service 3½ YEARS.

Disease BACILLARY DYS. Date of admission NOV. 27 1918 Date of discharge _____ Result CURED

Dates of Observation	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	1	2
Days of Disease	7																														
Temperature Fahrenheit	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time
Pulse per Minute																															
Respirations per Minute																															
Motions per 24 hours	38	32	24	16	13	4	4	3	2	3	1	3	3	2	4	3	2	2	2	1	1	1	1	2	2	1	1	1	1	1	1

BLOOD AND MUCUS + CHLORINE LAVAGE SERUM 100 CC CHLORINE LAVAGE DAILY B.A.D.M. MUCUS POSITIVE STOOL SERUM 80 CC CHLORINE LAVAGE SERUM 80 CC CHLORINE LAVAGE DAILY NEGATIVE STOOL CHLORINE LAVAGE DAILY MUCUS ABSENT SERUM 40 CC NEGATIVE STOOL NO MUCUS NEGATIVE CHLORINE LAVAGE EVERY 3RD DAY MOTIONS FORMED NEGATIVE NEGATIVE

(6378) Wt. W462/F506 2,000,000 2/18 McA & W Ltd A.F.B. 181/3 (E. 2565)

Signature _____ In charge of case.

CHART III.

EMETINE AND CHLORINE

CLINICAL CHART.
(To be attached to Case Sheet.)

Army Form B 181.

Corps _____ Military Hospital TEL EL KEBIR

No. 377 Rank and Name LIEUT T. C Age 33 (Service 3 YEARS) P.O.W. NO. 7.

Disease AMOEBIC Date of admission DEC. 8TH 1919 Date of discharge _____ Result CURED

Dates of Observation	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	1	2	3	4	5	6	7
Days of Disease	7																														
Temperature Fahrenheit	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time
Pulse per Minute																															
Respirations per Minute																															
Motions per 24 hours	28	26	17	15	15	12	9	9	9	6	6	5	4	3	4	3	3	3	3	2	3	3	2	1	2	1	2	2	1	1	1

POSITIVE AMOEBIC CHLORINE LAVAGE EMETINE GRS 1 POSITIVE CHLORINE LAVAGE EMETINE GRS 1 NEGATIVE NEGATIVE MOTIONS FORMED NEGATIVE NEGATIVE

BLOOD AND MUCUS + + MUCUS MUCUS MUCUS NO MUCUS NO MUCUS NO MUCUS

(6378) Wt. W462/F506 2,000,000 2/18 McA & W Ltd A.F.B. 181/3 (E. 2565)

Signature _____ In charge of case.

CHART IV.

shown to be of the greatest possible benefit to the patient. Evidence of this is shown in the study of *post mortem* material, and in the clinical results which are recorded.

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SILICOSIS.[1]

By H. E. McMahon, M.B., B.S. (Melbourne),
Kurri Kurri.

I have much pleasure in opening the discussion tonight on the subject of silicosis. I feel it is a great honour as this is our first clinical meeting. There are several present who could have handled the subject better, but, in accordance with your wishes, I have endeavoured to summarize the present knowledge on the subject, borrowing largely from the overseas authorities.

You are to be congratulated on taking up this question, as under our local conditions we have an almost unique opportunity for investigation.

Silicosis is one form of pneumonokoniosis (πνεύμων lung, κόνις dust). I find authorities spell this in four ways: pneumoconiosis (Osler), pneumonokoniosis (Tidy), pneumonoconiosis (*The British Medical Journal*), pneumokoniosis (often used, "Gould" uses it in discussing anthracosis).

Osler says:

> Under this term introduced by Zenker, are embraced those forms of fibrosis of the lung due to inhalation of dust in various occupations. The dust is inorganic or organic; the former being the more common and the more dangerous.

He classifies briefly: (i) Anthracosis, inhalation of coal dust and soot, lungs become carbonized; (ii) silicosis, from the dust of flint in small angular particles; (iii) chalicosis, from the dust of potteries, grinding steel *et cetera*; (iv) siderosis, from iron dust, red oxide of iron, brass and bronze.

Wynn writes:

> All the dangerous dusts have one common factor, they all contain free silica. Although the pulmonary disability induced by different dusts has received various names, miner's phthisis, grinder's rot, etc., the terminal pathological condition is always the same, and the victims die with advanced pulmonary tuberculosis. Although dust alone may produce disability and death, yet under modern social and industrial conditions silicosis really spells tuberculosis.

There are a number of questions we can discuss:

1. How does silica cause damage to the lung?
2. Is the action of silica modified by other dust, and is this modification just a matter of dilution, or do the adulterant dusts specifically inhibit or alter the action of the silica?
3. What action on the lung, if any, have non-siliceous dusts if inhaled over a considerable period of time?
4. How do the mortality and morbidity rates from lung diseases of coal miners compare with those of all occupied and retired males?
5. Where does the dust come from in the mines?
6. What are the dust conditions of our local mines, and what is the effect on the health of the workers?

Dr. Irvine, Chairman of the International Conference of Johannesburg in 1930, gives a good introduction to the subject of silicosis.

> The problem of what constitutes "silica risk" and of how it is to be countered is obviously fundamental since, if that can be solved, the pathology of silicosis becomes of no more and no less of special significance than that of any other disease, and the matter of compensation may be left to look after itself. The question of the ætiology and prevention of silicosis is plainly primarily a medical one, since without a well-informed and convincing lead from the medical side, the mining engineer or the industrialist must remain at a loss. As a world-famous engineer said at a recent Empire Mining Congress, "Let the physiologists tell us plainly what they want done, and we shall find ways of doing it for them". Hitherto the physiologists and other medical people have themselves been pursuing a painful process of self-education. Are we now in a position to do better? We know that certain dusts which contain free silica are phthisis-producing dusts, and that other dusts which

[1] Read at a meeting of the South Maitland Medical Association, November 23, 1932.

also contain free silica are not, and the accepted view at present is that the difference lies in the presence in the latter kinds of dust of other constituents, which nullify the harmful potentialities of silica. The facts are there, but their explanation is still somewhat obscure, although we can picture several possibilities.

Ætiology.

The first ætiological factor is exposure to silica dust, by which I mean dioxide of silicon in a free state, and not chemically combined in form of silicate, also concentration of the dust, and size of dust particles. (Particles must be under ten microns. Ten millions to cubic foot of 35% silica in quartz are tolerated over long periods without serious injury: Wynn.)

The second factor is the length of exposure. There has been a fatal result from tuberculosis, with evidence that it has been produced by silicosis after two and a half years' exposure (Middleton). Usually exposure is of many years' duration; it rarely occurs before four years, and often takes twenty or more years.

The third factor to be taken into account is the extent of the exposure period *plus* the latent period, and the relation of these to each other. Intermittency has a practical bearing.

The fourth factor embraces another important aspect, the influence of other dusts on the occurrence and course of silicosis. Other dusts may cause similar change; asbestos damages more rapidly than silica. Dr. Badham suggested that there was a silicatosis amongst the Sydney workers in blue metal quarries and certain metalliferous miners. Professor Cummins does not support this as far as his observations go.

The fifth factor is the physique of the worker. Race or age seems to have no effect. Pern suggests that sinusitis is a factor.

Pathology.

The Johannesburg International Conference of 1930 drew up the following:

The evidence obtained of the effects of the inhalation of silica dust in excessive quantities over prolonged periods of time shows that it has microscopically the following five stages.

1. A dry bronchiolitis characterized by an accumulation of dust-filled phagocytes in the terminal bronchioles, with desquamation of the epithelium.
2. The accumulation of dust-containing phagocytes about and in the intrapulmonary lymphoid tissue, and their transportation through the lymphatics into the tracheo-bronchial lymph nodes. (These two stages do not constitute the disease silicosis, the next three do.)
3. The gradual development of fibrous tissues within such accumulations of phagocytes and the formation of characteristic nodules of hyaline fibrous tissue.
4. Degenerative changes in these foci.
5. The hyaline nodules increase in size by extension at their periphery. Coalescence of adjacent nodules takes place and brings about involvement of further areas of the lung.

Macroscopically, in the early stage, a variable number of palpable pearly-white nodules up to two or three millimetres in diameter are found on the pleural surface of the lung. On section the cut surface of the lung is studded with pigmented foci, widely scattered, a moderate proportion of which are only just palpable. The tracheo-bronchial lymph nodes are slightly enlarged and deeply pigmented and may exhibit foci of fibrous induration.

In the later stages, the fibrotic nodules are increased in number, size and density, and coalescence of these may be found. The portion of the lung between the fibrotic nodules may be emphysematous. The tracheo-bronchial lymph nodes may be smaller in size than those in the early stage.

A combination of tuberculosis with silicosis may occur at any stage, and modifies the pathological picture profoundly.

Diagnosis.

For the diagnosis of silicosis as a disease it is necessary to take into consideration: (*a*) The employment history, (*b*) the symptoms and physical signs, (*c*) the radiological findings.

Clinically three stages of silicosis can be recognized.

In the first stage respiratory symptoms may be either slight or even absent. Capacity for work may be slightly impaired. There may be departure from normal in percussion and in auscultatory signs, and the radiograph must show an increased density of linear shadows, and the presence of discrete shadows, indicative of nodulation.

In the second stage there is increase in physical signs observable in the first stage, and the radiograph shows increase in number and size of the discrete shadows indicative of nodulation with a tendency to their confluence. There is some degree of definite impairment of working capacity.

In the third stage all the signs and symptoms are grossly accentuated, and indications of areas of massive fibrosis are usual, with serious or total incapacitation.

Pulmonary tuberculosis may be present in any of these stages of silicosis, altering the symptoms, physical signs and radiographic appearances and the degree of working capacity.[1]

Questions for Discussion.

After this general outline we can discuss our questions.

1. *How does Silica Cause Damage to the Lung?* Is it mechanical or chemical? That is, do the sharp particles damage the lung tissue, or is the action chemical from some portion dissolved in tissue fluids? The general trend of opinion is from the mechanical to the chemical. Osler and McCrae, 1920, speak of flint in small angular particles. Sir Harry Allen in "Pathology" says: "Silicious dust appears to act partly mechanically, partly chemically." Professor E. H. Kettle, in "Observations on the Pneumonoconioses" in *The British Medical Journal* of August 13, 1932, says: "It is, I think, generally agreed that silica is soluble in tissue fluids and that its action is, broadly speaking, chemical and not physical." All agree that the continued presence of

[1] At this stage Dr. McMahon showed a number of skiagrams and photographs showing the X ray appearances of the stages, and the changes with superimposed tuberculosis.

crystalline silica in the lung is followed by the gradual development of a characteristic fibrosis. Also, lately it has been shown that the silicate asbestos provides us with an example of dust other than crystalline silica, which is capable of producing similar lesions in the lung. Other silicates, kaolin for instance, are held to be entirely innocuous, as are also non-siliceous dusts. It is amazing the way an industrial worker can tolerate the presence of large quantities of foreign material in his lungs without apparently feeling any ill effects. The body can compensate in a marvellous way for the most remarkable structural derangements.

This is only the simple effects of dust on the lung, as Kettle says: "A much more important aspect of pneumonoconiosis is the influence of inhaled dusts on pulmonary infections, especially tuberculosis. Exactly how the silica influences the bacillus is not known. There is, however, much evidence to show that as it dissolves silica combines with protoplasm and converts it into a particularly favourable pabulum for the growth of the bacillus." The tissues are in some way altered to make them less resistant to the organism. It will light up a latent infection, or swing the balance in favour of the organism. How terrible the risk can be from unprotected working in silica is shown by the miners of twenty to thirty years ago, working with machine drills in the quartzite rock of the Rand mines.

Analysing the death certificates and histories of Cornish miners who returned, Haldane says:

> I found that of the many who died practically all had died of phthisis, and that the average duration of the work was 4·7 years, the average age at death being under 40. A few months of this sort of work without precautions seemed to mark a man for death sooner or later from phthisis.

2. *Is the Action of Silica Modified by Other Dust, and is this Modification just a Matter of Dilution, or do the Adulterant Dusts Specifically Inhibit or Alter the Action of the Silica?* That there is a modification is well shown by the analysis of figures of death rates from phthisis, for various occupations. I have not our local figures, but the English figures show this, as Haldane says:

> It will be seen that various occupations stand out in which a definite silicosis risk existed, while in others the phthisis risk is comparatively small, in spite of dust.

Among those with a high death rate from phthisis are workers in granite, sandstone miners, quarriers and masons, and pottery workers and grinders. Amongst those with a low phthisis rate in spite of dust inhalation are workers in igneous rock not true granite.

The figures for limestone quarriers and masons are much lower than for sandstone workers, but are still high as compared with those for coal miners.

> The relative immunity produced by coal dust is indicated by the low phthisis rate for coal boat loaders, as compared with the rather high rates for ordinary stevedores, and for other dock labourers. Cement working is one of the dustiest of occupations, the dust being a mixture of calcined lime and clay or shale; but the phthisis mortality is as low as that of coal miners.
>
> The fact that coal miners are, and have been for generations, less subject to phthisis than average persons is very striking, considering their conditions above ground. The coal dust and shale dust which they breathe are not antiseptic substances; but when we consider the physiological processes by which inhaled dust and bacteria are normally disposed of in the lungs, the relative immunity becomes intelligible. Inhaled dust-particles and bacteria are partly deposited on the walls of the respiratory passages and then swept upward by the action of the cilia lining these passages. Those which pass down farther to the lung alveoli are very quickly taken up by certain of the living cells lining the alveoli. These are the so-called phagocyte cells; and in accordance with the facts discovered by Metchnikoff, one of their functions is doubtless to digest and so destroy the bacteria. Their other function is to dispose of the insoluble dust; and this they do by casting loose and escaping upwards into the air-passages, or else into the lymph-passages. After an animal has been exposed to a cloud of coal dust or shale dust, these cells are found in abundance on their way up the bronchi; and the "black spit" of a coal miner is full of them. The lymph glands at the root of the lungs become also highly charged with them, and the final result is that if no further dust enters, the lungs are cleared of the inhaled dust.
>
> It is a well known fact that functional efficiency increases with use. Presumably, therefore, a collier's or a cement worker's lung becomes more efficient as regards its phagocytic activities than the lung of a person not exposed to dust; and this accounts for the relative immunity to phthisis of a coal miner or coal boat loader or cement worker.
>
> We must suppose that some soluble constituent in the dust stimulates the phagocytes to activity, and that this substance is present in coal dust, shale dust, clay, and various other kinds of stone dust which are not harmful.

This leads us up to our third question.

3. *What Action, if any, have Non-siliceous Dusts if Inhaled over a Considerable Time?* As Haldane says:

> It is surprising how much coal dust or shale dust or cement dust a man may breathe without apparent injury; but there must be a limit to this amount, so that exposure to very dusty atmospheres is most undesirable. When, moreover, a lung begins to be injured by bronchitis or emphysema, or by ordinary phthisis or silicosis, the capacity for dust elimination is necessarily impaired, so that local accumulations of dust occur. These seem to produce a scattered development of fibrous tissue giving an X ray picture similar to that produced in silicosis.

Professor Lyle Cummins points out that a recent series of investigations by medical men in South Wales has shown that the radiological appearances of lungs of coal miners after long service are in many cases similar to the appearances regarded as due to silicosis in gold miners and others exposed to silica dust. The pathological changes seen *post mortem* also reveal the same fibrotic changes in lung structure. Chemical analysis shows the same high silica content in the lung ash as has been noted in the silicotic lungs of the Rand miners and elsewhere.

As we have already mentioned, coal dust by itself is easily eliminated from the lung tissue, through the transportation in the interior of phagocytes, of the dust particles to points of exit or storage. Silica dust, on the other hand, tends to be retained in the lung tissue through its power of blocking up lymphatic channels and exciting the formation of fibrotic proliferation in the lung substance; and these drainage obstructing properties of silica interfere with the elimination from the lungs not only of the stone dust itself, but of the coal dust also. Cummins calls this a form of silicosis, "silico-anthracosis". By careful examination of the mortality records it is clear that the death rate due to phthisis of coal miners is much less than the average of all occupied and retired males. Why is this so? Professor Cummins replies:

> Many have thought that coal dust in itself has some antiseptic action and that prevents the occurrence of bacterial diseases by destroying bacteria inhaled into the lungs. The experiments of Wainwright and Nicholls, however, show that the germicidal

action of coal dust is practically nil; and it is obvious that the anti-infective action must depend on some factor other than a direct germicidal action. Experiments which I have carried out with my assistant Dr. Weatherall during the past year have shown that coal dust has a considerable power of adsorbing and rendering inert, the toxic principle of tuberculin—the toxic principle by means of which the tubercle bacillus appears to exert its lethal action on the tissues.

Coal dust acts as a factor in limiting the spread of the disease.

Haldane says:

The shale dust which had been breathed by coal miners in large amounts for generations, contained often about 30% of free silica, though coal miners had a remarkably low death rate from phthisis. Coal miners must breathe far more free silica than the Transvaal gold miners now did. When the free silica was mixed with the other constituents of shale schists, etc., it produced no phthisis.

4. *How do the Mortality and Morbidity Rates from Lung Diseases of Coal Miners Compare with Those of all Occupied and Retired Males?* I have not obtained our local figures, so will have to take the summary of the English figures used by the English authorities. We will compare those for pulmonary tuberculosis, pneumonia and bronchitis. We have already discussed the fact that the mortality rate from pulmonary tuberculosis is much lower than the average of the total population, but among coal miners in exceptional circumstances very great risk may occur among men driving roads through sandstone or other highly siliceous rock. "Some of the hard sandstone was found to contain about 82% of total silica and 71% of free silica."

We can discuss pneumonia in Haldane's words:

It has been suggested that the dust inhaled in coal mining causes, or may cause a high mortality rate from pneumonia. I think we may dismiss this suggestion. The mortality rate from pneumonia among coal miners in 1921–23 was only slightly higher than among all occupied males, but considerably less than for the social class of unskilled workers. This was so for all age-periods.

In regard to bronchitis mortality, it is generally agreed that there is a high mortality rate due to bronchitis in coal miners over forty-five years of age. As Professor Lyle Cummins says:

It is evident that the outstanding respiratory disease causing, or contributing to the mortality of South Wales miners is "bronchitis".

The facts are agreed to, but the authorities have differed somewhat in their reasons for this bronchitis. Professor Cummins says it is a straight-out "pneumoconiosis", essentially mechanical. In this he differs markedly from Professor Haldane's published statements. To the question "Why is the old coal miner so breathless?" he replies:

Because the lung alveoli, which should be clear and expansile, are to a great extent closed up or destroyed over large tracts of lung tissue through the accumulation of coal dust, the proliferation of fibrous tissue, and the destruction of air tubes and blood vessels, all the mechanical results from breathing in a mixture of stone dust and coal dust. Even in those parts of the lung which remain comparatively clear—and it is worth noting that the dust accumulations and the fibrosis tend to affect especially the apices of the lobes and the parts of the lung subjacent to the pleura—the mechanical effects are not wanting, for we find, in the least dusty parts of the tissue, a compensatory emphysema, a fusion and dilation of the alveoli, which diminishes the respiratory efficiency by decreasing the surface available for gaseous exchange. Doubtless, as pointed out by Haldane, the hard muscular work of coal mining plays a part in this latter change, but the factor of supreme importance in causing all these mechanical deteriorations is the retention of stone dust, and then coal dust, in the lung tissue, and the changes which these dusts produce in the lung structure.

Professor Haldane had said, in previous papers, that liability to bronchitis in later life depended on the amount of muscular exertion which an occupation implied. The bronchitis rate is seven times as great amongst those who work with their arms and legs, as amongst the so-called "upper" and "middle" classes. In his paper on the health of old colliers, he concluded that the essential cause of the unusual mortality from bronchitis among old colliers was the fact that their hard muscular work put an excessive strain on their lungs, leading to overstretching or emphysema, and the consequent shortness of breath, and liability to dangerous bronchitis. The strain during muscular work was immensely increased when, as used to be the case in coal mines, there was as much as 2% or 3% of carbon dioxide in the air. He now thought that the X ray evidence and the new facts brought forward by Professor Cummins showed distinctly that even the less harmful kinds of dust could produce serious pneumonokoniosis, and consequent disablement. New mining machinery, including conveyors, should be carefully considered from this point of view.

Cummins states that the essential cause of the breathlessness and bronchitis is "dust", Haldane says it plays a part, and should be controlled.

5. *Where Does Dust Come From in the Mines?* Dust comes from two sources: (*a*) From the coal and shales of the mine, (*b*) from stone dusting.

In regard to means of limiting the manufacture of dust, for miners working in sandstone or similar rock, to prevent inhalation of dust from machine drills, a dust trap similar to that introduced by Captain Hay should be used, and the heading should be well ventilated. No one should return after blasting until the place has been thoroughly cleared by ventilation. The broken rock should be wetted to prevent the raising of dust in shifting it.

It is of course impossible economically to prevent the formation of much of the coal dust in coal mines, but the following six points followed out will reduce it to a minimum.

1. Coal screening plant should never be placed nearer than eighty yards to the downcast pit, and the position of the screens should be so chosen that the prevailing winds blow the dust away from the shaft.

2. Skips should be so constructed that coal dust cannot fall through crevices on to the roads, and that they can be cleared of coal dust when emptied by the tipplers.

3. Skips should not be overfilled, as pieces of coal are liable to fall off and get crushed to dust on the haulage roads.

4. Keep all main roads and travelling roads free from small coal, because it is quickly ground into dust by the traffic of men and horses.

5. Avoid the use of high explosives, for by their shattering action they produce much dust, which finds its way into the returns; they also dislodge accumulated dust from the bars on roads in the vicinity.

6. Avoid fast-running haulages. Endless rope haulage at three miles an hour is sufficient.

Stone from Stone Dusting of Coal Mines.—Experiments carried out forty to fifty years ago showed that

coal dust suspended in air not only increases explosions, but is itself explosive. This danger could be greatly reduced by the addition to the air of an incombustible dust. Thus a 1 : 1 mixture of incombustible dust and coal dust is not ignitable in the presence of 4·5% or less of firedamp. The dust must be fine, at least 50% must pass through a 200 mesh sieve. Water can be used to prevent explosions, but has several disadvantages: (*a*) It is difficult to apply effectively, the finest dust particles are not kept down by it; (*b*) it has a bad effect on strata, causing it to creep and to disintegrate; (*c*) it raises the humidity, and so affects the working of the miners.

It is essential that the stone dust used must not contain too great a proportion of silica. Dr. R. Melville Hiley said he knew of one instance in which a roadway had actually been dusted, before a sample was submitted to examination. When this examination was carried out by a competent authority the "dust" turned out to be nearly pure oxide of silicon.

The injurious dust particles in mine air are very small, so fine indeed that they are invisible to the naked eye. Thus, air which appears pure may carry dangerous quantities of this very fine dust. The dust particles found in the lungs of miners who have succumbed to silicosis vary from ten microns in diameter to less than one micron. The number of dust particles contained in a lung in which there were twenty grammes of silica would be about six million millions, their average size being two microns. Dust above ten microns is considered harmless, as it is not stored in the tissue of the lung.

Men do not like working in a dusty atmosphere, so stone dusting is not popular; but the terrible loss of life in explosions in coal mines (in England there have been hundreds killed, and at Courrières, in France, there were over 1,100 killed in one explosion) forces preventive methods to be adopted, hence stone dusting is done. With the above in mind we can discuss our local conditions.

6. *What are the Dust Conditions of our Local Mines, and Effect on the Health of the Workers?* All the mines of our district have now adopted stone dusting, and are at present using limestone in which the free silica varies from about 0·5% to 7%. Only one shift is worked, and the dusting is done during the night; the new ground made is watered during the day.

In some mines the stone dusting is carried out enthusiastically, in others in a more perfunctory manner.

Since the aim in stone dusting is to get a 1 : 1 mixture with coal dust, it is necessary for the quantity of coal dust to be kept as small as possible. There could be great improvements in some of the skips used, as in many cases they have cracks and holes through which small coal freely drops on the haulage roads and is ground up. Besides, they are often overloaded, and toppers drop off frequently. Stanford Merthyr colliery had an explosion in 1904 in which six men were killed. At that time it was an open-light mine. Later they used water. In 1923 they commenced stone dusting with shale dust, and since 1930 have used limestone dust.

Pelaw Main colliery has dusted only since 1927, owing to the objection of the men to stone dusting. Since 1930 they have used limestone. Richmond Main colliery has been dusting since June, 1924, using shale, and limestone since December, 1930. The shale used was largely Borehole shale, up to 33% free silica.

In *Health* of May, 1931, is published the result of an investigation on fibrosis of the lungs in South Coast coal miners. The object was the determination of the incidence of fibrous pneumonokoniosis among coal miners before stone dusting had been in practice for any lengthy period—to prevent misconception as to the cause of pulmonary disease in these men.

The conclusion was:

That a fibrosis of the lungs may be caused by working in the coal mines of the South Coast, that the onset is slower and the disability less marked than the disease produced in metalliferous mines.

The practice of stone dusting with shale or limestone is only of recent origin, and has not been a factor in producing the fibrosis found.

In New South Wales we have three Acts: (i) The *Broken Hill Act* made for a special mining district. (ii) The *Workers' Compensation (Silicosis) Act*, which deals with Sydney sandstone workers. It grants up to £750, but nothing to dependants, except in case of death. (iii) The *Workers' Compensation Act*, 1926 (as amended), which grants compensation for fibrous pneumonokoniosis, except at Broken Hill. Diseases caused by silica dust were, however, excluded. Compensation payments are: maximum, £5 a week up to £1,000.

Case Histories.

I have a number of cases of chronic bronchitis and breathlessness amongst old miners; also a few cases in which the clinical and X ray diagnosis is pneumonokoniosis.

I will give three cases representing different types of histories.

Case I.—The patient has worked in coal mines only, in England and here. He worked last at Pelaw Main in January, 1928; he died in June, 1930, aged sixty-three years. He was paid compensation up to the time of his death for pneumonokoniosis, then full payment was made.

According to his history, he started work at eleven years of age in a coal mine in England. He came to Australia and worked for twenty-eight years at Minmi and Pelaw Main. He was chiefly on stone-work, development work, for fifteen years, shooting solid stone, a sandstone conglomerate, and he drilled the holes himself. He stated he breathed a lot of dust, although he wore a mask. He had dyspnœa, steadily becoming worse, and was losing weight, his respiratory excursions were very limited, expiratory breath sounds were prolonged over both lungs.

The X ray report in June, 1928, was that the examination showed an extensive mottling throughout both lungs. It was most marked in the mid portion and in the infraclavicular regions of both lungs. There were numerous calcified nodules scattered through both lungs. The appearance suggested advanced pneumonokoniosis with probably superimposed tuberculosis.

Case II.—The patient worked in gold mines and then in coal mines. He is fifty-five years of age. He has been working for many years in the coal mines here, but gives a history of working for some years in quartz mines. The X ray reports state that the appearances suggest pneumonokoniosis with superimposed tuberculosis. He is continuing working, but is losing weight and will ultimately be incapacitated.

Case III.—The patient is sixty-five years of age. He has a coal mining history for thirty years; before that he had a very

mixed industrial history; he was on a very dusty job in a coal mine. Clinical examination reveals dyspnœa, evidence of fibrosis of lung and chronic bronchitis. The X ray report is as follows:

> There is some slight yet definite enlargement of the heart shadow. The shape of heart suggests hypertrophy rather than dilatation. There is a very great increase in the lung markings, especially towards the bases, and a considerable increase in the hilar shadows. The appearances suggest chronic bronchitic changes with considerable fibrosis. There is some irregular "stippling" in both lungs and this with the fibrosis suggests the condition is pneumonokoniosis in the early second stage.

The part of the man's history that is interesting is the fact that since 1919 he has been working chiefly on brattice work—that is, pulling down the bagging which directs the flow of air. This stands for years, and is loaded with dust. The question is, is this constant inhalation of dust the cause of the pneumonokoniosis? The colliery is one in which the stone dusting is most thorough. He lost 6·3 kilograms (fourteen and a half pounds) during the past six months, but with a month off he has gained 1·8 kilograms (four pounds), feels better, and so resumed work again this week.

The X ray reports on these cases are all from Macquarie Street men, and entirely support the opinion of our local radiologist.

I have not found any tubercle bacilli in repeated examination of the sputum of these cases.

You will notice that I have not a case of pneumonokoniosis, either clinical or radiographic, in which the man has worked all his working life in our local mines.

We are only just commencing to investigate carefully the incidence of pneumonokoniosis and silicosis in our district. The final decision rests with the *post mortem* findings, and an analysis of the ash content of the lungs. At present all lungs of coal miners dying here are being sent for analysis.

It is on *post mortem* findings that Professor Cummins has based his silico-anthracosis theory, and it is by *post mortem* investigations amongst our local miners we will confirm or disprove his findings, and will know the effect on the lungs of the miners of the dust conditions in the mines.

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Reports of Cases.

A CASE OF PRIMARY GRANULOPENIA.[1]

By N. T. M. WIGG, M.B., B.S. (Adelaide), M.R.C.P. (London),
Adelaide.

THE following are a few brief details of a probable case of primary granulopenia. I follow the nomenclature suggested by THE MEDICAL JOURNAL OF AUSTRALIA, July 23, 1932.

Mrs. T., aged sixty-five, who had been semi-bedridden for many years with typical chronic rheumatoid arthritis, complained, on July 31, 1932, of a sore throat. For several days this was relatively mild, and her daughter, a trained nurse, did not deem it of sufficient severity to warrant medical attention until August 6.

On examination on that day the patient was slightly febrile, did not appear to be very ill, and the throat showed an intense redness of both tonsillar fossæ, spreading out into the adjacent fauces and soft palate. A diagnosis of streptococcal infection of the throat was made and the nurse asked to report and ordinary treatment prescribed.

On August 9 she was seen again, and was at once noticed to be much more seriously ill. Her temperature was 38·3° C. (101° F.) and her pulse relatively slow at 84. The fauces were more intensely red and inflamed though there was little swelling. The voice was slightly hoarse. A small patch of greyish-white membrane was beginning to appear in each tonsillar fossa. The patient also complained of pain in the vicinity of the anus, and on inspection an area of intensely red and inflamed skin was seen near the anal margin with necrosis commencing in the centre.

The following day, the tenth day of her illness, she was obviously worse, and examination revealed a spread of the faucial condition to the whole of the soft palate, with a patch of membrane on each tonsil about 1·25 cubic centimetres (half an inch) across. The temperature was 38·9° C. (102° F.) and the pulse rate 90. There was a definite jaundice of skin and conjunctivæ and bile was present in slight amount in the urine, which, however, contained no albumin. The anal condition had also extended and had a central area of necrosis. The stools were black and somewhat tarry.

The blood was examined for leucocyte count and films were made.

Later in the day she became semi-comatose and very restless. The temperature approached a hyperpyrexia, and death occurred some hours later.

I regret that *post mortem* evidence could not be obtained or that further investigation of the case was not possible.

The blood showed a leucopenia of 4,000. Dr. D. L. Barlow, who kindly examined the films for me, reported a complete absence of polymorphonuclear cells; he said that nearly all the cells seen were lymphocytes.

My reason for reporting this case is to stimulate interest in this curious condition. Admittedly, sufficient investigation was not made to prove this as a case of primary granulopenia, but the occurrence of an angina of the type described, associated with anal inflammation, an apparent complete lack of resistance on the part of the patient, death after a ten days' illness, with terminal jaundice, and a complete absence of polymorphonuclear cells in the blood, is typical of the condition. Piersol and Steinfeld, in the *Archives of Internal Medicine*, April, 1932, give this description as characteristic of the disease, and I was also interested by a paper by Damesaek and Ingalls in *The American Journal of the Medical Sciences*, April, 1931, page 502.

The condition is probably not a very rare one, although only about two hundred cases appear in the literature. As the prognosis is always so grave and treatment so ineffective, the value of knowledge of the condition would appear to rest more in putting the practitioner on his guard in anticipating such a condition in any person, especially a middle-aged or elderly female, already a sufferer from any debilitating condition, and, if possible, preventing its onset.

[1] Read at a meeting of the Section of Clinical Medicine of the South Australian Branch of the British Medical Association on August 17, 1932.

Reviews.

A BOOK FOR DIABETICS.

THE appearance of a second edition of "The Diabetic A B C" gives R. D. Lawrence an opportunity of bringing his well known line-ration scheme into conformity with

modern practice.[1] This is done by increasing the amount of carbohydrate contained in the line-ration; each line-ration now represents ten grammes of carbohydrate, instead of five grammes, as formerly. With this exception this useful little manual remains essentially the same as when it was first published in 1929.

Lawrence's line-ration scheme for the calculation and prescription of diabetic diets is widely and favourably known. All who make use of it in their practice or in their clinics will find the "Diabetic A B C" a most useful supplement to the verbal instructions which they are in the habit of giving to their patients. As its author writes, "it contains what I should like to teach every patient if I had enough time". Theoretical instruction is subordinated to practical advice; the field covered includes all the ordinary routine matters involved in the successful living of the diabetic life. In dietetic matters the line-ration scheme, of course, occupies the principal place. The explanation of this scheme is made additionally clear by a number of well chosen sample diets and recipes. The various aspects of insulin therapy are satisfactorily dealt with. The statement that "a certain dose (of insulin) burns and uses a certain amount of carbohydrate" is hardly in accordance with clinical experience; with this exception, however, the book contains little to blame and much to praise. Within its self-defined limits it will be found of considerable practical utility.

DIABETES OF CHILDREN.

Dr. Priscilla White has had the inestimable advantage of working for the past ten years in intimate association with five hundred and thirty-three diabetic children attending the Joslin Clinic. Much of the data gathered from the study of this wealth of clinical material is presented in "Diabetes in Childhood and Adolescence".[2]

Dr. Joslin has the knack of imparting his own statistical outlook on disease to all his fellow workers. As was to be expected, therefore, Dr. White's method of imparting knowledge is based very closely on that of her illustrious master. Perhaps the greatest service of the Joslin Clinic to the study of diabetes has been the clear and orderly manner in which the results of clinical investigation have been collated and set forth. The present monograph is in the best tradition of the clinic; clearness, brevity, and a refreshing lack of vague theorizing are its outstanding features.

Much of what is contained in the book is, of course, well known to all who work with diabetic children. The chapter on ætiology, however, breaks fresh ground. It contains the first really satisfactory statistical study on the hereditary factor in diabetes, and Dr. White draws conclusions from her data which are of the very first importance. It would appear, as had already been surmised, that the factor which governs the appearance of diabetes behaves as a Mendelian recessive, and that where the family history of two partners to a marriage is known, the likelihood of diabetes appearing in their offspring can be calculated with almost mathematical accuracy. In the case of the mating of two diabetics, the probability is that all of the children will develop diabetes; where one partner to the marriage is diabetic and the other has a diabetic family taint, 50% of the children will be so affected; where neither partner is diabetic, but both have a family taint, 25% is the calculated figure; finally, where one partner is diabetic or has a diabetic taint, but the other partner has an absolutely clear family history, it is in the highest degree unlikely that diabetes will ever develop in the offspring of such a marriage. These calculations are based upon a sufficiently large series of cases to be reasonably reliable. They cannot but prove of the greatest assistance to all who are called upon to advise on the problems presented by the marriage of diabetics.

In calculating diets for diabetic children, Dr. White has definitely adopted the policy of giving reasonably large amounts of carbohydrates; no child ever receives less than 100 grammes of carbohydrate *per diem*. Diets are, however, rigidly measured; in the author's experience it is the diabetic whose diet is lax who is most frequently subject to those distressing complications, arteriosclerosis, cataract and coma. It is impossible to expect that every worker in this field will agree in detail with all Dr. White's recommendations; the onus, however, is on dissentients to produce better results in their own patients than those produced by the method with which they disagree. It would be hard indeed to present a more successful record of treatment than is contained in this book.

Chapters on growth and development, on complications, on diabetes in infancy, and on coma have each their particular interest; a record of one death in seventy cases of coma treated since 1923 must surely be unique. It certainly sets a standard for the rest of the world to follow.

The book is well printed, and the ninety-eight statistical tables which it contains are clearly set forth. A few regrettable misprints will doubtless disappear in subsequent editions. Alike to the diabetician and to the general physician, the book will be found invaluable.

NURSING AND DISEASES OF THE NERVOUS SYSTEM.

A series of conversational clinical lectures by an eloquent speaker may be delightfully instructive to those interested, but much may be lost when they are published in book form. In "Nursing in Nervous Diseases", by Dr. McConnell, the author does not attempt to cover the whole field of nervous diseases.[1] Perhaps it is a pity, as there are relatively few books (apart from books on mental diseases) which treat this branch of nursing with due importance.

One would expect the book to be essentially practical. An opening chapter on the nervous system, introducing the "central lobe" and the "*pons oblongata*" (terms somewhat confusing to the British student), is supported by anatomical diagrams labelled extravagantly beyond the contents of the text. "Nomenclature" and "The Neurological Tray" are but necessary evils in a specialized subject, and the chapters are pleasingly concise. On "physical therapeutics" the author is so delightfully vague that one wonders whether something may not emerge from the electrical boxes illustrated in the best advertising style. Various neurological conditions (which are not put forward as complete) are described briefly for the nurse, but, while useful hints are made with regard to details, the nurse will require to refer regularly to her "General Nursing" to fill in the many gaps.

In the later chapters much space is devoted to the qualifications for the ideal nurse, more particularly in the treatment of the neuroses and milder psychoses, all of which are discussed in the short space of one chapter. The nurse may object to being described as one "who is probably a human being", and may refuse to accept as trained one who takes the "hypersensitive young lady a tray on which the napkin was soiled with spilled soup" *et cetera*.

[1] "The Diabetic A B C: A Practical Book for Patients and Nurses", by R. D. Lawrence, M.A., M.D., F.R.C.P.; Second Edition; 1932. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 63. Price: 3s. 6d. net.

[2] "Diabetes in Childhood and Adolescence", by P. White, M.D., with foreword by E. P. Joslin, M.D.; 1932. Philadelphia: Lea and Febiger. Royal 8vo., pp. 249, with 25 engravings and a coloured plate. Price: $3.75 net.

[1] "Nursing in Nervous Diseases", by J. W. McConnell, M.D.; 1932. Philadelphia: F. A. Davis Company. Demy 8vo., pp. 153, with 24 engravings. Price: $1.50 net.

The Medical Journal of Australia

SATURDAY, APRIL 1, 1933.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE FEDERAL COUNCIL.

At its recent meeting in Melbourne the Federal Committee took the final step towards the inauguration of the Federal Council of the British Medical Association in Australia. The Memorandum and Articles of Association of the Federal Council were signed preparatory to its incorporation. The Federal Committee, in the ordinary course of events, will not meet again. The inauguration of the Federal Committee was the most important event in the history of the Australian Branches; its passing is a sign of their progress and a witness to the wisdom of those who first sought their unity. Those present at the signing of the Memorandum and Articles of Association of the Federal Council could not do other than call to mind William Thornborough Hayward, George Adlington Syme, and Robert Henry Todd, men of vision, statesmen of medicine, who by their inspiration, their devotion and their unselfishness broke down prejudice, to replace it by mutual trust, and who welded the medical profession of the Commonwealth into a united body to fulfil its destiny. It is fitting that at this stage in the history of the Australian Branches we should pay homage to their memory.

The statement has often been made that the Federal Committee has outlived its usefulness. Though this may be partly true, members of the Branches will do well to consider its achievements. The harmonious relations and the mutual trust of the Branches are no myth. The old interstate jealousy is a thing of the past. Members visit States other than their own for scientific discussion, and Branches refer to one another questions affecting medical practice in a way which would have been impossible before the Federal Committee came into being. Two State journals, *The Australasian Medical Gazette* and *The Australian Medical Journal*, have given place to one journal which serves the whole of Australia. The Australasian Medical Congresses have been brought under the ægis of the British Medical Association. The Federal Committee has been the connecting link between the parent Association and the Branches; it has been instrumental in making for the Branches necessary alterations in constitutions and ethical procedure, matters requiring the approval of the Parent Body. It has been the mouthpiece of the medical profession when action or cooperation of governments has been needed. It has done all these and other things, and done them well; and its actions have not been made easier either by the Great War or by the financial depression.

The chief handicap of the Federal Committee was that it had no power to initiate new movements or to institute reforms; it dealt only with matters brought before it by the Branches. The Federal Council which takes the place of the Federal Committee will have wider powers. The medical profession will therefore expect more from it. If the Federal Committee failed to deal with large questions affecting the whole of Australia, it was the fault of the Branches for not setting the machinery in motion. If the Federal Council fails to give a lead in similar matters, the Branches and their members will hold it responsible. At its first meeting the Federal Council will have to deal with two important subjects. It will receive a report from a special subcommittee on the inauguration of a so-called representative body, a convention, for the six Australian Branches. It will also consider a report on the formulation of a hospital policy for the Branches. There is no need to discuss either of these matters at the moment, but it is obvious

that something must be done in regard to both of them. Reference has been made to those who stood out as leaders in the past. It is easy to say that we have not a Hayward, a Syme or a Todd, but it must be remembered that the need produces the man. Members of the medical profession need not doubt that their leaders will rise to the occasion. Should the leaders fail, the remedy is in the hands of the Branches; they are the masters of the Federal Council.

Current Comment.

HEALTH AND CLIMATE.

The relation of climate to health and energy is a matter of daily discussion by people everywhere; yet, despite much scientific investigation, as well as centuries of experience, man understands very little of the physiological and pathological effects of climate. Some observers believe that a tropical climate takes toll of the bodily and mental health and vigour; others hold the view that improper living conditions and mode of living, bad hygiene and sanitation, and endemic transmissible diseases, and not the climate *per se*, are the causes of the excessive invalidity and loss of energy associated with life in a tropical country. Certainly, as more is learnt of the ætiology of so-called tropical diseases, fewer and fewer are ascribed to tropical climate. Still, there is strong evidence to show that climate has an influence on the human organism, as it has on lower forms of life. For many years the health-giving properties of sunlight have been recognized, and within recent years considerable progress has been made towards an understanding of the mode of action of sunlight. It is known that extremes of atmospheric heat and cold may cause profound metabolic disturbances and even death. It is only rational to assume that less pronounced changes of temperature, humidity and barometric pressure should affect metabolism. In a recent paper Kenneth Black discusses the effects of climate on health and energy, with special reference to Malaya.[1] Black remarks that little is known to medical science of the various phenomena that determine climate—atmospheric temperature, wind, seasons, moisture, storms, sunshine *et cetera*—yet climate is probably one of the most important factors in the life of man; it has a direct influence on health as well as an indirect influence through food, incidence of disease, and mode of life. Man can live wherever he can obtain food and drink, but his physical and mental energy and his normal character are developed to their full only when he is adapted perfectly to his environment. To illustrate his point Black uses Huntington's map, on which about half the United States of America and a portion of Western Europe are shown as the only areas in which human health and energy are at their highest; most of the tropics is marked "very low", much smaller parts are shown as "low" and "medium". Huntington deals rather unkindly with Australia and New Zealand; he marks Victoria, Tasmania, and the extreme south of the North Island and the whole of the South Island of New Zealand as "high" (not "very high"); part of New South Wales, a small area of southern Queensland, and a narrow strip of the southern part of the Australian continent are labelled "medium"; the central part of the continent, from the east to western coasts, is marked "low", and practically the whole of the territory north of the Tropic of Capricorn is marked "very low". Huntington obtained his information concerning health and energy by means of a *questionnaire*; the reliability of the information might thus be doubted. Possibly one reason why Australia is pictured as having a comparatively low standard of health is that it was less known than many other countries to Huntington and those to whom his questions were directed. Cilento has pointed out that if the areas inhabited by less than two people per square mile and the endemic areas of hookworm disease or malaria or both are marked on a map of the world, the remaining areas will be found to coincide fairly closely with Huntington's areas of "very high" health and energy. In Australia the areas free of hookworm disease and malaria are roughly those marked "medium" and "high" on Huntington's map. Notwithstanding the objections mentioned above, even when allowance is made for the unavoidable inaccuracies, it must be agreed that Huntington's map contains some evidence of a climatic distribution of healthy and unhealthy conditions.

Black remarks that the white man can colonize a tropical country, but only at the expense of his efficiency. Europeans who have remained in the tropics for long periods without a break deteriorate greatly in mind and body; regular vacations spent in an optimum climate, such as that of Western Europe, are necessary. Garlick found that the area surveyed each month by white men making a topographical survey of Johre increased rapidly when each man was becoming accustomed to the work and conditions, then remained at about the one level for less than two and a half years, when it slowly decreased, until the end of the third year, when the men were relieved. Garlick remarked that "this drop was unquestionably due to the strain of working in a climate in which the shade temperature was seldom below 80° F., day or night, winter or summer". Black discusses at some length the mental effects of long residence in a tropical climate; he lays particular stress on the frequent occurrence of neurasthenia and the incidence of suicides in Malaya and other tropical countries.

Of course, the obvious flaw in the arguments of persons who blame the tropical climate for the ill-health or loss of efficiency of tropical residents is that they fail to take sufficient account of the

[1] *The Malayan Medical Journal*, December, 1932.

influence of poor food, lack of entertainment, poor living conditions, loneliness, the society of natives of poor intelligence and unhygienic habits, the absence of home life, minor illnesses, more serious disease, and the thousand and one other factors that make life in the tropics entirely different from life elsewhere. There is no means of estimating the importance of these factors; until there is, the increase in knowledge concerning the influence of climate on human health and efficiency is likely to be slight. As the years go by and more is learnt of mental function and bodily economy and disease, the more mature views of competent observers will be interesting. For the present the logical view is that climate, general conditions and diseases all play their part in the causation of ill-health and loss of energy in the tropics. Though the importance of each varies with localities and individuals, disease is the most important factor. The personal factor is difficult to estimate and more difficult to control; every medical practitioner in the tropics comes in contact with people who are quite unsuited to a tropical life, and others who seem to flourish in spite of all difficulties and disadvantages. If it were possible by undertaking an extensive general survey to determine the personal qualities necessary to successful and happy life in the tropics, some constructive work might be done.

BACTERIOPHAGE.

Bacteriophage has been the subject of much investigation during the years that have passed since d'Herelle, following the lead of Twort, made his striking announcements regarding it, yet there is still considerable divergence of opinion concerning its therapeutic value. Mention of the subject has been made before in these columns, and reports of the work of Burnet, McKie and Wood have appeared in this journal. An important finding by these workers was that the administration of a polyvalent Flexner bacteriophage had no influence on the course of infantile dysentery. Other workers have had similar disappointing results; but others again are convinced of the value of bacteriophage in the treatment of bacillary dysenteric affections. In India particularly, encouraging results have been obtained. A remarkable feature revealed by the investigations is the great number of types or races of bacteriophage active against a particular organism. The study of the bacteriophages and their properties has become very complex. In any experiment many factors other than the mere presence of bacteriophage must be considered. A failure to appreciate this may have been the cause of discrepancies in some of the earlier work.

An important contribution to the study of bacteriophages has recently been provided by J. Morison, who has been particularly interested in cholera in India.[1] Morison remarks that Asheshov was the first to show that, for the effective action of bacteriophage in infective diseases, as well as the production of complete and permanent lysis *in vitro*, the bacteriophage must be polyvalent, or rather, "multi-type". Bacteria cultured with a single type bacteriophage rapidly become resistant to the particular type. Appreciation of this phenomenon enables the investigator to type any particular race of bacteriophage. For example: let it be assumed that there are three types (*A*, *B* and *C*) of bacteriophage active against a particular organism; it is possible to obtain three cultures of this organism resistant to *A*, *B* and *C* types of bacteriophage respectively; the culture resistant to *A* is lysed by *B* and *C*, that resistant to *B* is lysed by *A* and *C*, and so on; organisms resistant to an unknown bacteriophage can thus be tested against known types. Investigations along these lines have revealed interesting information; for example, it has been found that races that have been cultured separately and mixtures consisting of races that have been cultured separately are unstable, whereas natural mixtures remain stable and potent for at least two years at room temperature on the plains of India. This phenomenon may account for many of the failures to confirm d'Herelle's findings. Morison points out that d'Herelle used natural mixtures.

Morison remarks that in Assam the most important centres for cholera are the banks of the "dying" rivers. The Kalang River may be taken as an example. It is an old silted bed of the Brahmaputra, which it leaves at Silghat and rejoins, after winding ninety miles through a flat alluvial plain, above Ganhati. During the winter the natives use the dry mud flats of the Kalang as latrines; in the warm weather, when the waters of the Brahmaputra rise and flow into the Kalang, flooding the mud flats, cholera spreads along the Kalang Valley. In such a place adequate measures of sewage disposal are at present impracticable. Some other means of controlling cholera must therefore be devised. Vaccination would be adequate; but it has many disadvantages, and has not proved a great success in native communities. Morison conceived the idea of issuing supplies of mixed cholera and dysentery bacteriophage to each village headman, to be administered to every person suspected of suffering from cholera. The experiment was carried out in the Nowgong district, on the banks of the Kalang River; the result was that no epidemic occurred during 1930 and 1931. In the district of Habiganj, another "dying" river district, no bacteriophage was administered, and the usual spring and autumn epidemics of cholera occurred. The object of the administration of bacteriophage in an insanitary area is, of course, not only the immunization of the person treated, but also, through the polluted water supply, the remainder of the community.

As yet no report on the incidence of cholera in 1932 is at hand. It will be interesting to note whether the Nowgong area has remained free, although occupied by a population unprotected by vaccination or recent outbreaks.

[1] "Bacteriophage in the Treatment and Prevention of Cholera", by J. Morison, C.I.E., M.B., D.P.H.; 1932. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 32, with illustrations. Price: 4s. net.

Abstracts from Current Medical Literature.

DERMATOLOGY.

Insulin Dermatitis.

SCHENK (*Deutsche Medizinische Wochenschrift*, October 7, 1932) reports two cases of insulin dermatitis occurring in diabetics whose condition was of five and seven years' standing. The ætiology may be either an anaphylaxis against the insulin itself or possibly to the protein contents of the preparation. In both the cases reported by the author the protein content of the insulin was the cause. Treatment consisted of changing the brand of insulin, desensitizing the patient with small intracutaneous doses of insulin, and giving calcium by mouth and by intravenous injection.

Pruritus Treated by Histamine.

A. C. ERNSTENE AND B. M. BANKS (*The Journal of the American Medical Association*, February 4, 1933) furnish a preliminary report on the use of histamine in the treatment of pruritus. They gave histamine subcutaneously, usually in doses of 0·5 milligramme twice a day, to six patients suffering from pruritus associated with urticaria and to seven patients with pruritus due to other conditions. Three of the six patients whose pruritus was accompanied by urticaria were promptly relieved. One of these three patients obtained complete and lasting relief from itching. In the other two a relapse occurred. One of these, on further treatment with histamine, manifested considerable improvement, and in the other the treatment was only partially effective. Four of the seven patients with pruritus due to conditions other than urticaria manifested improvement. Of these three, one suffered from *pruritus vulvæ* of three years' duration; another suffered from pruritus in the crural regions of uncertain ætiology; a third had generalized dermatitis of unknown origin, and the fourth had *kraurosis vulvæ*. The three patients in this group who received no benefit from histamine all suffered from *pruritus ani*. The authors hold that the results of their study warrant further investigation of the value of histamine in the treatment of pruritus.

Recovery from Pemphigus Foliaceus.

J. F. SMITH (*The British Journal of Dermatology and Syphilis*, December, 1932) reports a case of recovery from *pemphigus foliaceus*. The patient was a male, aged forty-two years. He was spare and almost emaciated. His whole skin surface was involved by typical *pemphigus foliaceus*. Nikolsky's sign was strongly present. The heart, lungs, abdomen and nervous system were apparently normal. The Wassermann test gave no reaction. After temporary improvement following the use of saline baths and soft paraffin dressings his condition steadily deteriorated. He had fever, his skin was very foul-smelling and large areas were denuded. He looked almost moribund. He was given tryparsamide by intravenous injection in weekly doses of one gramme, and later on of two grammes. Within four months he had recovered completely. The author points out that recovery from *pemphigus foliaceus* is very rare.

Callosities, Corns and Warts.

ARTHUR WHITFIELD (*The British Journal of Dermatology and Syphilis*, December, 1932) discusses the developments of callosities, corns and warts. Callosities are physiological hypertrophies, developed as a protection against pressure and friction where the subcutaneous bone is that normally present. They occur alone and are also found, almost invariably, surrounding tender lesions such as warts and corns. A corn gives rise to pain even when the overlying pressure is removed. Within the area of the callosity surrounding a corn are one or more points differing in colour from that of the callosity. These points are whitish and are due to irregular and somewhat parakeratotic cornification combined with the presence of air between the layers. On microscopic examination a corn is found to be a peg-shaped mass of irregular keratinization extending much lower in the epidermis than that of the surrounding callosity. The author regards as quite erroneous the commonly believed statement that a bursa is usually present beneath a corn. He believes that a corn develops in a situation where there is pressure, but also where the subjacent bone is either not normal or is not normally underlying the skin. In other words, where there is either inflammatory deformity of the bone or in the presence of a subluxation of a joint, the latter being by far the more common. A wart is a papillomatous tumour. It is formed of thickened mucous and horny layers in varying relative proportion, overlying hypertrophied papillæ containing enlarged and dilated blood vessels. Though warts are commonly infective, the author holds that they are likely to develop without infection over traumatic telangiectases.

Mercurial Inunctions in Syphilis.

H. N. COLE and others (*Archives of Dermatology and Syphilology*, January, 1933) have investigated the excretion of mercury following the use of mild mercurous chloride inunctions. They point out that mercuric chloride is the basis of many preparations planned to replace the unclean and irritation-producing mercurial ointments of earlier days. They also add that there is great variance of opinion regarding the value of mercurous chloride inunctions. They carried out experiments with six male patients who had no eruption of the skin. Four grammes of a 50% mild mercurous chloride ointment were rubbed into the skin every day for thirty days. One patient salivated on the fourteenth day; this was regarded as being due to an idiosyncrasy. The amount of mercury excreted in the urine rose slowly and progressively and reached a maximum of 0·09 milligramme per day at the end of the fourth week. The amount excreted in the fæces rose rapidly to 0·45 milligramme per day at the end of the first week; the level remained almost constant during the second and third weeks and then declined but slightly to the end of the fourth week. The authors compare these results with those obtained by the use of 5% metallic mercury inunctions; in these circumstances the urinary excretion is 0·15 milligramme at the end of four weeks, and the fæcal excretion 0·25 milligramme. The authors explain the low excretion of mercury in the urine after the use of mercurous chloride inunctions by the difficulty of rubbing the hard particles of mercurous chloride into the hair follicles; absorption is for this reason poor.

UROLOGY.

Serial Pyelography.

T. D. MOORE (*Journal of Urology*, October, 1932) emphasizes the value of taking three exposures on one film side by side, within a space of about thirty seconds, during the course of excretion urography. By this means the dynamic state of the pelvis and ureter at different moments is observed, so that the muscular movements of the walls of these channels may be noted. Moreover, if the second of the three exposures is made in deep inspiration while the other two are at the end of expiration, the respiratory excursion of the kidney is clearly visible. Filling defects, regions of spasm and the relation of suspected shadows to the urinary channels are conveniently noted and their pathological significance evaluated by the serial method.

Pyelography in Renal Tuberculosis.

E. SIMON (*Zeitschrift für Urologie*, September, 1932) comes to the following conclusions regarding the diagnostic value of cystoscopic and excretion urography in renal tuberculosis: (i) The pyelogram of surgical renal tuberculosis is quite characteristic and is easily distinguishable from that of the ordinary pyonephrosis, except when destruction in a tuberculous pyonephrosis is so advanced as to wipe out all differences. (ii) With modern methods and non-irritating contrast media cystoscopic pyelography should be quite harmless in renal tuberculosis, but the necessity for its use has been largely obviated by the introduction of excretion urography. (iii) Patients do not come to the surgeon till clinical symptoms appear, and by then the pathological changes are so advanced that the excretion pyelogram enables the surgeon to complete the diagnosis. In doubtful cases cystoscopic inspection,

with cystoscopic pyelography added if necessary, will complete the examination. (iv) When an excretion shadow is lacking on one side and the clinical symptoms and urine examination indicate urinary tuberculosis, the indication for removal of a non-functioning kidney is undoubted, provided the excretion pyelogram on the opposite side is normal.

Dynamism of the Prostate.

I. MIHALOVICI (*Journal d'Urologie*, August, 1932) states that the pyeloscopic study of renal and ureteric dynamism initiated by Legueu and his co-workers in Paris has stimulated study of the dynamics of other parts of the genito-urinary tract. The present study concerns the prostate gland, whose functions are threefold: (i) motion, (ii) secretory, and (iii) nervous. The motor function is that of expelling prostatic fluid during ejaculation. Erection of the *colliculus seminalis* helps the intimate mixing of the seminal and prostatic fluids. The secretory function is internal as well as external. The nervous functions are subserved as follows. The secretory nerves run from the inferior mesenteric ganglion down through the hypogastric nerves. The motor nerves, from the pelvic plexus, provoke contraction and emptying of the gland. Sensation of the ordinary desire to micturate arises from cerebral perception of undulating muscular contractions of the bladder wall, when this organ is adequately filled, but imperious desire to urinate has its seat of sensation in the nerve endings of the posterior urethra and the prostate gland. These endings resemble tactile Pacinian corpuscles. It is also likely that the prostate is a peripheral nervous centre for the provocation of erection. Affections of the dynamism of the prostate are as follows: (i) Spasms or colic of the prostatic musculature causing severe pain, referred to the perineal, ural, rectal, low back and lower limb areas. (ii) Functional retention of secretion without mechanical obstacle. (These retentions follow on atony of the repressing musculature. The atony arises from various causes.) (iii) Incontinence of the secretion resulting from syhincteric atony. There are no sphincters, properly so called, at the mouths of the prostatic ducts; the prostatic muscle fibres are simply thickened as they coat the terminal portions of the ducts. Prostatorrhœa is the chief clinical sign of the adynamic prostate. As treatment, massage and irrigations are of prime importance in the adynamic cases, with faradization added if necessary, using both urethral and rectal electrodes.

Aneurysm of the Renal Artery.

M. GERARD (*Journal d'Urologie*, November and December, 1932) has made a study of renal artery aneurysms. This condition is rather rare, but in recent years at least one new case is published each year. The diagnosis is hardly ever made *ante mortem*, chiefly because the possibility of its occurrence is not considered. The author strongly deprecates use of the term "false aneurysm" for the condition of perirenal hæmatoma following rupture of the renal arteries in renal contusions. There exists but one variety of renal artery aneurysm, true aneurysm, and to this condition he limits his study. Sixty-one cases have been traced in the literature, and of these twelve have been deleted as being "false" aneurysms. From a careful study of the remainder the author comes to the conclusion that the classic symptomatological triad of pain, hæmaturia and perirenal tumefaction is false. This triad was characteristic of false aneurysm and was applied by extension to the true forms. Aneurysms of the renal artery are encountered about equally in men and women. They are nearly always unilateral and single, and occur about equally on either side. The actual site is very constant; they occur almost always outside the renal parenchyma, just within or just without the hilum, and are intimately connected with the pelvis. For reasons of clarity in exposition, the author has been obliged to separate non-ruptured renal artery aneurysms from the cases of actual rupture. Those which do not rupture usually develop in aged subjects and have arteriosclerosis as their principal cause. They are of slow development and tend to become calcified. Their prognosis is relatively benign. Those which rupture are caused by factors which ordinarily tend to aneurysm and they develop chiefly in young subjects. Their evolution is insidious without clinical manifestations till rupture occurs. Their prognosis is grave. In the benign group some pain in the renal area is common and trifling change may be present in the urine. If these signs lead to a urological examination, an annular shadow may be noticed in the region of the hilum in the plain radiogram; pyelography shows that this shadow is outside the pelvis. In the dangerous group the accident of rupture is revealed clinically in three distinct ways: (*a*) The hæmaturic group comprises one-third of the cases; they are accompanied by pains in the renal area. (*b*) The group with perirenal tumefaction is the most frequent and comprises about one-half of the cases. (*c*) The mixed form, a combination of (*a*) and (*b*), is rare, and is the only group fulfilling the description of the old classic syndrome.

Gonococcal Urethritis.

I. MIHALOVICI (*Zeitschrift für Urologie*, September, 1932) states that one of the most important moments in the course of an acute gonococcal urethritis is that at which the onset of infection of the posterior urethra occurs, as is evidenced by a previously clear second test glass becoming turbid. At this time the patient notices a tendency to a somewhat increased frequency of urination. In the management of the acute anterior urethritis rather weak potassium permanganate (one part in 10,000) irrigations of the anterior urethra have been given. The author continues these after the onset of posterior infection, but at once commences a course of intravenous urotropin injections. He has found that the best results are obtained by giving a dose of 1·25 grammes each day for at least five days. A 25% solution in distilled water is used; therefore five cubic centimetres will be injected at a time. The most important point is to give the first injection as soon as the second test glass becomes hazy. By this means infection of the prostate and seminal vesicles can be prevented in most cases and the duration of treatment considerably shortened.

The Leucocytic Formula in Renal Tuberculosis.

D. NOVACEF (*Journal d'Urologie*, November, 1932) describes the state of the leucocytes in different stages of renal tuberculosis. In all forms of the disease there is a hyperleucocytosis and an increase in the number of neutrophile cells with undivided nuclei. In the early stages of the disease the normal relative proportion of neutrophile cells, mononuclear cells and eosinophile cells is undisturbed. In advanced cases there is a hyperneutrophilia. In chronic cases with poor resistance the mononuclear cells and eosinophile cells are increased in number.

Uretero-Peritoneal Fistula.

G. L. HUNNER AND H. S. EVERETT (*Journal of Urology*, September, 1932) report a further case of urinary ascites due to uretero-peritoneal fistula. The patient was treated for carcinoma of the cervix by radium and deep X ray therapy. After a long and stormy convalescence her condition improved considerably, but she had a vesico-vaginal fistula. Nine months later she was readmitted to hospital with an abdominal tumour and anuria. After four days she passed urine spontaneously through the fistula, which admitted the index finger. *Paracentesis abdominis* produced sterile urine. Bilateral ureterosigmoid anastomosis was performed, about three litres of urine being evacuated from the peritoneal cavity. There was no evidence of carcinoma and above the pelvis the peritoneum was smooth and glistening, but the sigmoid, rectum and uterus were densely adherent. The operation was exceedingly difficult and prolonged, and the patient died eight days later. The authors direct attention to the extraordinary tolerance of the peritoneum to urine, and also the late effects of irradiation by massive doses on the deeper tissues. They found much necrotic tissue about the rectum and uterus in this case. They suggest that patients who are subjected to deep therapy within the pelvis should report back every six months for investigation of ureteral function, as it may be seriously jeopardized by either the disease or its treatment.

Special Articles on Treatment.

(Contributed by Request.)

V.

CARBUNCLE.

Carbuncle is a localized infective gangrene of the subcutaneous tissues. It results from the infection of the dense subcutaneous fat by a pyogenic bacterium, of which the usual type is *Staphylococcus aureus*. The pathway of infection is either direct from the cutaneous surface by way of hair follicles or by direct injury, or by the blood stream in cases of auto-infection. The condition does not usually occur in those who are in perfect health, but rather in those persons whose resistance is lowered by debility, often resulting from *diabetes mellitus*, glycosuria or nephritis. A brief consideration of the pathology is necessary for a proper appreciation of the rational treatment. Locally, the carbuncle occurs usually in an area which already has a poor blood supply. As a result of inflammation and infection a thrombo-phlebitis develops and the local blood supply is cut off. This precipitates local gangrene so that a slough of the subcutaneous fat occurs. The overlying skin shares in this event, but the deeper muscle only rarely suffers. On account of thrombosis at the periphery of the inflamed area, and the indifferent blood supply of fat, the separation of the slough is very tedious, and subsequent healing by granulations is distressingly delayed. From the infection present the slough may partially melt down into a thick pus, which is extruded through many openings in the overlying skin. It is very important to remember that the vessels at the edge of the carbuncle, in the brawny œdematous area, contain infected clots which may be readily dislodged into the general blood stream by improper squeezing, thus producing pyæmia, or in the case of the face and neck, intracranial sinus infection, with fatal effects. Constitutionally, the outstanding pathological condition is the profound toxæmia that may result, or even pyæmia or septicæmia.

As in all surgical affections, it is well to subdivide the treatment into that of the local condition and that of the general or constitutional effects.

Local Treatment.

The objectives of the treatment of the carbuncle itself are: (i) to localize the condition and to prevent its further extension, (ii) to secure the earliest possible removal of all the necrotic material, (iii) to relieve pain, (iv) to secure rapid epithelialization after the slough has separated.

Preliminary Treatment.

Immediately the diagnosis has been made, the affected part should be put at absolute rest, because every movement causes pain. If the carbuncle should happen to be located on an extremity, the limb must be placed in splints. The next step is to secure the cleanliness of the area. The skin must be lightly shaved, washed thoroughly with soft soap and warm water, and then cleaned with ether or alcohol. As a temporary measure the application of generous hot saline fomentations is permissible. The fomentations should be changed at least every hour, unless necessary movement of the patient distresses him too much.

Surgical Treatment.

There are two distinct schools of surgical treatment. These are the non-operative and the operative.

Non-Operative or Conservative Treatment.

Non-operative or conservative treatment is incomparably the best in the following circumstances: (*a*) In all cases in which localisation has taken place, (*b*) in all cases in which there is a large area of surrounding brawny induration, (*c*) in all carbuncles on the face. If there is no definite evidence of the presence of an actual slough, it is permissible to try the effect of hot fomentations, Bier's hyperæmia, cupping or watchful expectancy.

As soon as there is clinical evidence of gangrene, either with or without a break in the continuity of the epithelial surface, treatment should become more energetic, though still conservative.

Masterly Inactivity.—There appears to be a very distinct swing of opinion in the direction of masterly inactivity. It is realized that there is a very dangerous zone of infected thrombosed vessels at the periphery of the carbuncle, from which clots may readily be displaced into the general circulation. Also, the separation of the slough, though tedious, is most efficiently performed by the natural mechanism. The advantage of this procedure is safety. The disadvantages are important, inasmuch as the patient is asked to suffer pain and discomfort for a longer period, he is incapacitated for a period which may or may not be of longer duration, and the amount of toxic absorption from large carbuncles may perhaps be greater. Masterly inactivity does not imply absolute neglect, but rather attention directed to complete local and general rest, as well as intelligent combating of the general condition.

Magnesium Sulphate Fomentations.—Prepare a saturated or, rather, a supersaturated solution of magnesium sulphate. Have the solution at a temperature of about 48·8° C. (120° F.) and wring out sterile lint soaked in this fluid. Apply these fomentations at least every two hours, whether the surface is broken or intact. The results achieved from this method are excellent. It is entirely devoid of danger. The solution is so hygroscopic that a constant flow of lymph containing healthy phagocytes occurs in the inflamed region. This occurrence favours the removal of local toxins and the earlier removal of the slough. The magnesium sulphate method is especially valuable in the treatment of carbuncles situated on the lips or elsewhere on the face.

Magnesium Sulphate plus Glycerine Compresses.—Use as external applications pieces of sterile lint soaked in a saturated solution of magnesium sulphate to which has been added glycerine, five ounces to the pint. This method is an extension of the last named and may have some advantages inasmuch as the applications have not to be changed so often.

Magnesium Sulphate Paste.—The use of magnesium sulphate paste was introduced by the late A. E. Morrison. A paste is prepared as follows: Place in a hot mortar eleven ounces of *glycerinum acidi carbolici* and add one and a half pounds of dried magnesium sulphate. The paste is slowly stirred and mixed with a warm pestle until the result is a thick white cream. This paste is exceedingly hydroscopic and must not be left exposed to the air, because it would rapidly become a fluid, and useless. The paste must be kept in a covered jar. The paste is applied as a thick layer on a piece of lint and then placed directly upon the carbuncle and the nearby inflamed area. The lint should be generously covered with cotton wool and the dressing changed about twice in the twenty-four hours. It is found that this paste produces a liberal flow of healthy lymph, so that the separation of the slough is greatly accelerated. From a moderate sized carbuncle the slough will probably separate within a week. The application of this paste quickly alleviates pain in the lesion and causes considerable improvement of the constitutional condition. When the slough has come away, the raw surface or the granulating area may still be treated with this paste, or perhaps better by the application of gauze soaked in supersaturated solution of magnesium sulphate. The magnesium sulphate and glycerine paste method is excellent. It cannot be recommended too strongly. It is the method of choice in all carbuncles of the face and in all cases in which grave constitutional disorder renders the giving of an anæsthetic or operation inadvisable.

Operative Treatment.

Immediate operation upon all carbuncles was once the widely accepted treatment, but now it has lost favour somewhat, except in special cases. Operation is indicated when the general condition is so grave that the patient

is not likely to withstand a long toxæmia and when the slough is unduly long in separating. Do not operate in cases of carbuncle of the face, nor in those that are probably streptococcal in origin.

Pre-Operative Preparation.—Very little is called for in the way of preliminary preparation. An enema may be given, but the urine should always be collected and tested for the presence of glucose. If glucose be found, energetic anti-diabetic measures are at once instituted, such as the injection of a suitable dose of insulin. The skin over and around the carbuncle is cleaned with ether or spirituous biniodide solution. The use of iodine or any of the staining antiseptics should be avoided, as they confuse the clear definition of the extent of the inflamed area. An ordinary ether anæsthetic given by the open method is quite suitable.

Excision of the Carbuncle.—Some surgeons believe in the total extirpation of the carbuncular area by a wide circular excision. This has the advantage that it at once removes the site of toxin production and hence improves the general condition. The disadvantages are naturally that it is applicable to lesions of reasonable size only, that the resultant raw surface is extensive and has to be covered by plastic surgery or skin grafts, or a large area is left to heal by granulations.

Incision of the Carbuncle.—Incision of the carbuncle is much more usually adopted. A deep incision is made to the full depth of the carbuncle and extends across its full diameter. Then another similar incision is made at right angles to the first, both crossing at the centre of the carbuncle. Four quadrants are thus produced. Some surgeons are content with this procedure and then apply concentrated magnesium sulphate foments, waiting until the sloughs separate without artificial aid. Others adopt a more radical attack and, raising each flap in turn, cut away all necrotic tissue until a fresh oozing surface is exposed both at the floor of the carbuncle and underneath each flap. Excessive bleeding may be controlled by pressure, the application of hot saline pads, or by the cautery. The resultant wound is packed with gauze saturated with magnesium sulphate and glycerine solution, or gauze smeared with the paste mentioned above. The dressing will be changed daily.

Curettage of the Necrotic Tissue.—Without incision necrotic tissue may be removed by a curette or a spoon which is introduced through the cribriform openings on the skin surface. This method is to be condemned, as it is sure to open up infective venous channels and may cause systemic dissemination of organisms.

Cauterization with Carbolic Acid.—Cauterization with carbolic acid is a secondary rather than a primary procedure and is sometimes carried out after removal of the necrotic tissue, whether by block removal or by curettage. The raw surface is swabbed over by gauze soaked in strong carbolic acid solution. The cavity is afterwards lightly packed with plain gauze. This method is hæmostatic and antibacterial as well as productive of some local anæsthesia, but there is a certain amount of risk of increasing the necrotic area as well as producing a general carbolic acid poisoning from absorption.

Post-Operative Care.—Keep the skin around the carbuncle well swabbed with alcohol so as to prevent infection of other hair follicles, with resultant boils. Gauze packing should be removed daily and the wound cleansed with saline solution, hydrogen peroxide (eight volumes), or eusol. Hydrogen peroxide is very suitable. Carefully repack underneath the flaps so as to secure steady, regular granulation of the cavity. Encourage epithelial growth by the use of red lotion. If there is a very large area left for healing by granulation, the best procedure is to cover the area by some form of skin grafting.

General Treatment.

The objectives of the constitutional treatment of carbuncle are: (i) to eliminate toxins and combat the toxæmia, (ii) to promote the body resistance to infection, (iii) to promote rest, sleep and alleviate distress.

In the first instance it is essential to discover the presence, if any, of an underlying medical malady, such as *diabetes mellitus*, glycosuria, nephritis, or focal sepsis. These must at once receive appropriate energetic treatment. The patient is confined to his bed, and this period may extend for many weeks. Fresh air and even sunlight or direct sun exposure is desirable. The diet must be generous, light but nourishing; eggs and milk are valuable; brandy and other alcoholic drinks that the patient is in the habit of taking may be permitted. Fluids may be given in large quantities; barley water, water, lemonade and imperial drink are suitable. External hydrotherapy by means of sponging or even baths is very helpful. The elimination of toxins by the bowels must be attended to by giving calomel, say, 0·12 gramme (two grains) every second or third day, and saline aperients are very satisfactory. The production of sweating by blankets helps to remove toxins. Experiment with the usual hypnotics, to find one that suits the patient, since adequate sleep is absolutely essential in these cases. The use of morphine is quite justifiable and, indeed, may be imperative if, owing to pain and distress, the patient cannot get sufficient rest. Hypodermic injections of 0·015 gramme (one-quarter of a grain) of morphine should suffice. Vaccine therapy is disappointing and may be omitted. Blood transfusions at the beginning of treatment may be valuable, especially in debilitated and asthenic patients. During convalescence tonics should be given.

Conclusions.

1. Simultaneous attention to the local and general condition is essential.
2. Carbuncle must be regarded as a very serious complaint, and one in which a localized infection may readily be converted into a systemic infection.
3. Carbuncles must not be roughly handled or squeezed, and this especially applies to those on the face.
4. Conservative local treatment gives the best results. The concentrated magnesium sulphate applications give very excellent results.
5. Masterly "neglect" is often surprisingly successful.
6. Prevent the spread of boils by keeping the surrounding skin frequently swabbed with ether or spirit.
7. Hesitate before adopting radical operative measures.
8. Treatment is long and arduous, so preserve the patient's strength and morale.
9. Frequently test the urine for albumin and sugar.

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British Medical Association News.

SCIENTIFIC.

A meeting of the Victorian Branch of the British Medical Association was held at the Alfred Hospital, Melbourne, on September 21, 1932. The meeting took the form of a series of clinical demonstrations by members of the honorary staff.

Intraocular Foreign Body.

Dr. J. Ringland Anderson and Dr. W. M. Box showed a patient whose eye had been pierced by a splinter of steel from a ploughshare. Nine months after the injury ophthalmoscopic examination revealed the presence of the foreign body. Localization was carried out according to Gonin's method for the localization of retinal tears. A Foster-Moore stud was introduced and was observed to be close to the foreign body. Removal of the foreign body by means of a magnet was simple. There was siderosis, but the patient had recovered his visual acuity to $^{9}/_{12}$.

Detachment of the Retina.

Dr. Anderson and Dr. Box also showed two patients who had been successfully treated by Gonin's methods for detachment of the retina. In one case the detachment was of long standing and there was extensive retinal degeneration. In the other case the condition was due to an anterior retinal dialysis. The patient was a boy, aged sixteen years; he had fully recovered his vision.

Alcohol Amblyopia.

Dr. Anderson and Dr. Box next showed a patient who had suffered from toxic amblyopia due to alcoholism for a period of three years. There had been sudden severe loss of vision. No improvement had occurred. The main loss was in the form of central scotoma.

Bilateral Retrobulbar Neuritis.

The next patient shown by Dr. Anderson and Dr. Box was a girl, aged six years, suffering from bilateral retrobulbar neuritis, probably due to nasal or tonsillar sepsis. Vision had been recovered.

Cerebral Tumour.

Dr. Anderson and Dr. Box also showed two patients suffering from cerebral tumour. One of the patients was also suffering from syphilis. The other was a boy who had papilœdema and visual fields suggestive of a chiasmal lesion. There was no evidence of any pituitary dysfunction. The skiagrams demonstrated the changes associated with internal hydrocephalus. At operation a large cerebellar astrocytoma was found. The visual loss and the alteration in the visual fields were thought to have been due to the internal hydrocephalus.

The visual fields of a patient suffering from cerebral tumour which had been operated upon and which had eventually recurred, together with the *post mortem* findings, were also demonstrated.

Cerebral Abscess.

The next patient shown by Dr. Anderson and Dr. Box was a boy suffering from optic atrophy. He was shown with the object of emphasizing the value of perimetry when there was any suspicion of a temporo-sphenoidal abscess complicating mastoiditis. He had developed hemiplegia, aphasia and seizures, which had commenced in the contralateral shoulder, with deviation of the head and eyes away from the side of the injury.

Charts of visual fields from another patient were shown to demonstrate an homonymous hemianopia of cortical type due to an occipital abscess. This abscess was secondary to a cervical cellulitis. The fields were the only localizing sign. The abscess had been opened and drained, but death had ensued.

Squint.

Dr. T. A'B. Travers showed a series of patients suffering from squint with abnormal retinal correspondence. The retinal anomaly was shown to have a varying degree of persistence under treatment. In some cases the anomaly was overcome with orthoptic training, in others by operation, and in others the anomaly appeared unchanged, although the eyes had been put straight by operation years previously.

Cerebral Syphilis.

Dr. R. S. Ellery showed four patients suffering from neuro-syphilis, illustrating the clinical types and response to treatment. The first, affected with *paresis sine parese*, was a male, aged fifty-one years, who had come to hospital with delusions of grotesque type. He had been wandering about the streets, was mildly disorientated and slightly confused. Examination of the cerebro-spinal fluid revealed pleocytosis and excess of globulin. The cerebro-spinal fluid reacted to the Wassermann test (+40). The condition yielded to treatment by "Tryparsamide". The mental condition cleared up completely. At the time of the meeting there was no increase of cells nor excess of globulin in the cerebro-spinal fluid, and the Wassermann reaction was positive (+10).

Another case was that of a typical exalted paretic in the early acute stage. He underwent treatment by malaria and made a perfect recovery. At the time of the meeting he had been back at work for over three years and was playing in a symphony orchestra. He was still attending hospital for anti-specific treatment.

Two other patients, males, aged forty-eight and twenty-eight years respectively, exhibited the classical signs of tabo-paresis. Both responded satisfactorily to treatment by "Tryparsamide", and the reactions of their cerebro-spinal fluid were becoming less pronounced. Both patients had been under intensive treatment for eighteen months.

The lesson to be learned was that "Tryparsamide" must be used over fairly lengthy periods to get the best results; that there was no parallel between the clinical and the serological response to the drug; that optic atrophy was not a contraindication to its use, but that every patient should have his fields examined before embarking upon a course of injections; and that the initial doses should be small, say, from 0·5 gramme working up to 3·0 grammes a week. The course of injections should last from eight to twelve weeks, and the patient should rest between each course for at least a month. Intramuscular injections of bismuth or mercury might be usefully administered in the rest period.

Arterial Embolism.

Dr. Paul G. Dane showed a middle-aged man, suffering from aphasia of the verbal type, due to an arterial embolism affecting the pre-central region of the brain. The patient was suffering from mitral stenosis, and had previously had an attack in which he became almost blind and giddy. When first seen in February, 1932, he was quite speechless, and could not communicate in any way. At the time of the meeting he was able to read a newspaper, and to speak a few words quietly and answer a few simple questions.

Psycho-Neurosis.

Dr. Dane next showed five patients suffering from psycho-neurotic disorders. Four of the patients were women; the fifth was a young man. They had all been treated by hypnotic suggestion for some weeks and complete relief from symptoms had been provided in all cases but one.

The first patient was a married woman who had had four children and was fearful of further pregnancy. This had led to complete sexual anæsthesia, accompanied by many morbid thoughts, insomnia and terrifying dreams. These symptoms had all disappeared and she was now quite well.

The second patient was a young girl, suffering during the previous few years from attacks of vaso-vagal epilepsy. Every day she would have at least six attacks, and was quite unable to do her work. By the time of the meeting she had returned to work, and suffered only from an occasional attack. Her sleep and general health had also improved.

The third patient was a married woman, who had been confined to bed for three months with a functional paraplegia, and for a further four months she had only been able to stagger about. She was subject to recurring spasms of the muscles of the back, which would cause her to fall. All these symptoms had been completely relieved, and she was in better health than she had been for years.

The fourth patient was a young girl who had been compelled to leave her work, owing to restlessness, inability to concentrate, and obsessional fears that she would throw herself out of trains or trams. She was completely relieved and had returned to work.

The fifth patient was a young man who for years had suffered from lack of confidence and extreme nervousness, incapacitating him from all work and enjoyment. He was now completely relieved.

Asthma.

Dr. Charles Sutherland showed two patients suffering from asthma. The first was a man, aged forty years, who had had asthma for eighteen years. Tests with pollens, foods, feathers and other special forms of dust caused no

reaction, nor did his history suggest extrinsic factors as a cause of his symptoms. There was a faint reaction to the von Pirquet test; there was complete achlorhydria. Treatment with acid was not at all helpful, and vaccines, peptone and special diets did not help. Operation on the nose for polypus aggravated the attacks for some months. In October, 1930, treatment with tuberculin was started, and the asthma had gradually lessened in severity, although mild attacks still occurred.

For comparison Dr. Dane showed a man who used to get severe attacks in the early summer but who was always free in the winter. By skin tests sensitiveness to grass pollens was revealed; for the past two summers, following desensitization, no attacks had occurred.

Hay Fever.

Dr. Dane's next patient was a boy, aged eleven years, who had had attacks of sneezing all his life. Examination of his nose revealed changes typical of vasomotor rhinitis. His skin reacted markedly to egg white and to several grass pollens. He had avoided egg and had been desensitized to pollens and had improved.

Another boy, aged fourteen years, suffering from vasomotor rhinitis, was shown. He had reacted only to feathers and "house dust". He had greatly improved since desensitization to these substances.

Eczema.

Dr. Dane's last patient was a boy aged five years, who had had eczema since the age of fourteen months. The disorder was always worse in the summer. He had been breast fed up to the age of five months. Skin tests revealed severe reactions to pollens and rather less severe to horse dander, egg white and mustard. Avoidance of the foods indicated, and desensitization to the pollens concerned had improved the condition greatly.

The methods used in testing with pollens and foods were demonstrated and the technique of desensitization was discussed in detail.

Pathology of the Gall-Bladder.

DR. R. A. WILLIS showed a series of specimens illustrating various aspects of gall-bladder pathology. The first exhibit was a series of unusual specimens of gall-stones, the second a series of specimens of carcinoma of the gall-bladder, the third comprised examples of *cholecystitis cystica*, and the fourth was a series of specimens illustrating the part played by phagocytic, lipoid or foam cells in biliary pathology.

Suppurative Conditions of the Knee Joint.

DR. BALCOMBE QUICK showed three patients to illustrate the capacity of repair inherent in the knee joint in suppurative conditions under conservative treatment.

The first patient was a boy, aged fourteen years, who had received a severe blow upon the front of the knee on August 18, 1932. He said that a few drops of clear fluid had oozed out. Some twenty-four hours later the joint had become swollen and very painful and he was unable to move it. He was admitted to hospital on August 20, 1932. His temperature was then 37·8° C. (100° F.), his pulse rate was 120 and his respiration rate 28 per minute. The joint was swollen, red, hot, and painful on the slightest movement. An abrasion was present at the upper margin of the patella. Tenderness was pronounced lateral to the quadriceps tendon. The leg was fixed in a Thomas's splint with light extension. On August 22, 1932, offensive blood-stained fluid was aspirated. *Bacillus welchii* and *Streptococcus viridans* were found in the aspirated fluid. On August 23, 1932, 10,000 units of gas gangrene antiserum were given. This was repeated the following day. On August 25, 1932, the joint was washed free of offensive clot by irrigation with Dakins' solution and hydrogen peroxide, exit being given by incision over the medial aspect of the joint, which was left unsutured. The temperature remained moderately elevated for the following fortnight, after which it subsided and there was no further cause for anxiety.

The next patient was a boy, aged fourteen years, who had been admitted to hospital on October 16, 1931. Eighteen weeks before he had been operated upon in the country for acute osteomyelitis of the tibia. Six weeks before admission a sequestrectomy had been carried out. This had been followed three weeks later by an acute suppurative arthritis, which had been treated by lateral incisions with rubber dam drainage down to the synovial membrane.

At the time of admission the temperature was 38·4° C. (101·2° F.) and the pulse rate was 120 per minute. There was no pain, but the knee was extremely swollen, flexed almost to a right angle and with a range of movement of 30°. X ray examination revealed destruction of the medial portion of the head of the tibia, including the articular surface, with absorption of joint cartilage. Fixation and light extension were employed, and the temperature subsided and the leg became straight. Premature active movement resulted in a rigor and a rise of temperature to 38·9° C. (102° F.), which subsided in six days. At the time of the meeting the patient had been walking without crutches for two months, having a painless knee joint which possessed a full range of movement, although some grating could be felt.

The third patient was a woman, aged thirty years, who had been admitted to hospital on October 8, 1930. Four months before the left knee had suddenly become swollen and painful. The pain was excruciating on any movement. The pain and swelling had been increasing. For a week there had been aching pain in the left calf. For two days there had been shivering attacks. The temperature was 39·5° C. (103·2° F.), the pulse rate 136 and the respiration rate 36 per minute. The knee was swollen generally, and there was tenderness in the lower part of the popliteal space. Four years previously the patient had had a miscarriage followed by sepsis of the left leg and pneumonia.

Pus containing *Staphylococcus aureus* was aspirated from the knee joint, and a small amount of similar pus was evacuated from beneath the medial head of the gastrocnemius muscle by incision in the popliteal space. X ray examination revealed a central abscess in the patella. The anterior wall of the cavity was freely removed and a valvular communication with the joint through the articular cartilage was demonstrated. The joint was irrigated with a solution of "Flavine" and the abscess cavity packed with gauze soaked in bismuth-iodoform-paraffin paste, which was allowed to remain undisturbed for a fortnight, the joint meanwhile being immobilized. Some fifteen months later an abscess developed in the left thigh. Except for this the patient had enjoyed good health and the function of the joint was perfectly normal.

Papillomata of the Anal Region.

Dr. Quick also showed a male patient, aged twenty-four years, who had suffered from an ischio-rectal abscess two years previously; this had burst and a warty growth had developed in the affected region. This mass, said to have been the size of a mandarin, had been excised three months prior to the meeting; recurrent growths had been excised a month later. Examination of a histological section showed papillomata upon a chronic inflammatory base. There was no reaction to the Wassermann test, and the past history was one of uninterrupted good health. A soft papillomatous mass, the size of a pullet's egg, was seen in the scar. Two superficial fistulæ in relationship to this mass were lined with similar papillary growth. Suggestions were asked for regarding treatment, and it was agreed that diathermic excision was probably the best method of attack.

Acromio-Clavicular Dislocation.

Dr. Quick's next patient was a man, aged twenty-three years, who had been operated on for an old acromio-clavicular dislocation. Prior to operation some five weeks earlier, abduction of the arm could not be carried above the horizontal, and weight carrying was not possible. At operation the acromio-clavicular ligaments had been

restored by fascial grafts passed through drill-holes in the bone ends. Full functional restoration had resulted.

Cardiospasm.

Dr. Quick also showed a man, aged twenty-eight years, who had had difficulty in swallowing for two years and a feeling of obstruction in the epigastrium. He had had attacks of dull pain beneath the sternum, and sometimes vomited undigested food. He had been treated elsewhere by bougie dilatation without relief. X ray examination confirmed the diagnosis of cardiospasm and showed a "fairly marked dilatation of the œsophagus".

At operation a year prior to the meeting the stomach had been opened and digital dilatation of the cardiac opening carried out for several minutes, three fingers being introduced. Since then there had been no further vomiting, almost all the lost weight (eight kilograms or eighteen pounds) had been made up, and the patient expressed himself as almost completely relieved. Recent X ray examination showed that the œsophagus was no longer dilated.

Hallux Valgus.

Dr. Quick next showed two patients who had suffered from double *hallux valgus* and had been treated by operation with satisfactory results. The operation was directed almost entirely to the phalanx, of which the proximal half was removed. Any osteophytic outgrowths were chiselled off the metatarsal head, but none of the articular surface was removed. The shortening of the toe thus brought about effected a relative lengthening of the extensor tendon and the shortened lateral ligament.

Compound Fracture of the Leg.

Dr. Quick's last patient was a male, aged fifty years, shown to demonstrate the great utility of the Sinclair modification of the Chutro stirrup in securing extension in a difficult case of compound fracture of the leg. The stirrup, which was passed, under local anæsthesia, through the space in front of the *tendo Achillis* and above the *os calcis*, maintained a painless and effective extension for as long as necessary. Foot-drop was automatically corrected and there was no tendency for stiffness to develop at the ankle joint.

(To be continued.)

Congresses.

THE THIRD INTERNATIONAL PÆDIATRIC CONGRESS.

The Third International Pædiatric Congress will take place in London on July 20, 21 and 22, 1933, and will be held in the central part of London (the exact location will be announced later). The President is Professor G. F. Still, the Treasurer is Dr. Hugh Thursfield, and the Secretary is Dr. Leonard Findlay.

Members of a recognized medical society are eligible for membership of the Congress, but they must be nominated by their own national committee.

The subscription for medical men or women (active members) attending the Congress is two pounds sterling (or two pounds and ten shillings to include a copy of the transactions), and for non-medical individuals accompanying active members one pound sterling. All subscriptions are payable in advance to the Treasurer, Dr. Hugh Thursfield, 84, Wimpole Street, London, W.1.

There will be two sessions daily, from 10 to 13 o'clock in the morning and from 14.30 to 16.30 in the afternoon.

Two of the morning sessions will be devoted to general discussions. The following two subjects have been selected for discussion: (a) "The Nature of Allergy and Its Rôle in Diseases of Children", which will be introduced by Professor Hamburger, Vienna, Dr. Pehu, Lyon, Dr. Arnold Rich, Baltimore; (b) "The Prophylaxis of Milk-Borne Diseases", which will be introduced by Professor Allaria, Torino, Professor Bessau, Berlin, Professor Lereboullet, Paris, Professor Pettersson, Stockholm.

Nominations are invited from the secretaries of the various national committees of delegates to take part in the discussion of these subjects. Copies of the communications of the introducers will be available one month before the meeting, and requests for these should be made to the Secretary of the Congress.

Delegates desiring to make communications should do so through their own National Committee, but it must be understood that the final decision regarding any communication rests with the British National Committee. The titles of papers to be submitted should be sent to the Secretary of the Congress (Dr. Leonard Findlay, 61, Harley Street, London, W.1) as soon as possible, and the manuscript, ready for publication, not later than May 23, 1933. Further information may be obtained from Dr. J. W. Grieve, 12, Collins Street, Melbourne.

Post-Graduate Work.

POST-GRADUATE COURSE IN ADELAIDE.

The next post-graduate course arranged by the Adelaide Permanent Post-Graduate Committee will be held in Adelaide during the week commencing May 22, 1933. The following is a provisional syllabus of the course. The final syllabus will be forwarded to intending members shortly before the course begins.

May 22, 1933.

Dr. F. H. Beare: A demonstration of common neurological conditions.
Dr. H. R. Pomroy: "Complications Following the Operation for Acute Appendicitis, and Their Treatment."
Dr. Alan Hobbs: "The Treatment of Fractures Near the Elbow and Wrist."
The E. C. Stirling Lecture, by Dr. H. Douglas Stephens, of Melbourne: "Limp in Infancy and Childhood."

May 23, 1933.

Dr. Guy Lendon: "Diagnosis and Management of Dyspepsias of Gastric Origin" (subject to alteration).
Dr. G. H. Burnell: "The Treatment of Acute Gonorrhœa in the Male, and Its Complications."
Dr. I. B. Jose: "Modern Methods of Managing Cases of Prostatic Enlargement."
The E. C. Stirling Lecture, by Dr. H. Douglas Stephens, of Melbourne: "Acute Abdominal Conditions in Infants and Children."

May 24, 1933.

Dr. Thorold Grant: A demonstration of medical diseases in children.
Dr. L. A. Wilson: "The Treatment of Acute Pleural Empyema."
Dr. J. S. Verco: A demonstration of radiological technique in common conditions.

May 25, 1933.

Dr. W. Ray: A clinical demonstration in the medical wards.
Dr. Gilbert Brown and his assistant anæsthetists: "Premedication and Basal Narcosis in Anæsthesia."
The Listerian Oration (under the auspices of the South Australian Branch of the British Medical Association).

May 26, 1933.

Dr. T. G. Wilson and Dr. R. F. Matters: A demonstration of various obstetrical cases and their management.
Dr. B. H. Swift: A demonstration of gynæcological cases and their treatment.

May 27, 1933.

Dr. D. R. W. Cowan: Clinical demonstration in the medical wards.

The Joint Honorary Secretaries are Dr. Allan Lamphee and Dr. Leonard Lindon, both of 188, North Terrace, Adelaide. The Committee will be grateful if intending members would notify either of the secretaries or the Secretary of the South Australian Branch of the British Medical Association at 206, North Terrace, as early as possible of their intention to be present. It is hoped that arrangements will be made whereby members may go into residence at the Adelaide Hospital during the week following the course. The fees will be as before, namely, two guineas for attendance at the whole course and one guinea for those desirous of attending the E. C. Stirling Lectures only.

LECTURES IN SYDNEY.

THE New South Wales Permanent Post-Graduate Committee announces that four lectures on new principles in physiology and their application will be given by Professor W. A. Osborne, of the University of Melbourne, at the Robert H. Todd Assembly Hall, British Medical Association Building, 135, Macquarie Street, Sydney, concurrently with the post-graduate course, from May 29 to June 9, 1933, at 8.15 p.m.

Tuesday, May 30: "The New Dynamics of the Circulation."

Thursday, June 1: "Metabolism and Repair of the Connective Tissues. Normal and Pathological Functioning of Smooth Muscle."

Tuesday, June 6: "The Normal and the Surgical Stomach."

Thursday, June 8: "The Solution of Some Engineering Problems in Medicine."

The fee for the course will be two guineas or one guinea for those who are resident medical officers at hospitals. The lectures are open to all medical practitioners. The names of those wishing to attend should be sent to the Honorary Secretary, New South Wales Permanent Post-Graduate Committee, 235, Macquarie Street, Sydney.

Obituary.

EDWARD PETER LEAVY.

DR. EDWARD PETER LEAVY, whose death was recently recorded in these pages, was born at Dublin on July 2, 1856. He began his education at a Christian Brothers' school at Dublin, and in 1886 began to study medicine at the Royal College of Surgeons of Ireland. He qualified in due course and held resident appointments in Ireland. Seeing no opening in the land of his birth, he came to Australia, where he became associated with Dr. Birmingham at Charters Towers. Two years later he went to Geraldtown, known at present as Innisfail. Here he held the appointment of Medical Officer to the Innisfail Hospital for a time and for many years acted as Government Medical Officer. He established his practice on a sound basis and did his best to keep abreast of advances in medicine. At the age of fifty-nine he returned to his native land and qualified for the Fellowship of the Royal College of Surgeons of Ireland. He was a great reader and was devoted to music; for some years he had sung professionally as a tenor. He interested himself in sugar-growing and owned a small plantation. He was married twice; he is survived by his widow and a daughter born of the first marriage.

GERALD GEORGE KELLY.

WE regret to announce the death of Dr. Gerald George Kelly, which occurred on March 23, 1933, at Brisbane, Queensland.

Correspondence.

AN ECONOMIC COMMENTARY AND DIAGNOSIS.

SIR: May I answer my critics? I will take them in order. I agree with Dr. Springthorpe that we need "Health of Body, Truth of Mind, and Good Will of Spirit", but think that we have much of these now, and would have more had we an economic system which permitted us to express and develop them. As people are said to have the government they deserve, so they probably have the economic system they deserve: our dissatisfaction with the results of the present one insures a change, so that we need to find out in what way our national economy can encourage freedom, justice, brotherliness among our people. It will take all kinds of minds to attain such a grand aim as a free, noble and virile people.

The "Cobbler" accuses me of knavery or folly: knavery I deny, but if wisdom consists in anonymity and folly in signing one's name, then folly I cannot deny. Pensions, charities and gaols were bracketed as different modes of providing for the unfit, whether unfit from age, ill-health, poverty, misfortune or crime. Surely a just economic system which will prevent most of these ills is preferable to ours where sentiment is called upon to repair the effects of injustice.

Dr. Trinca accuses me of being an amateur economist, a charge more correctly brought against those economists who are content to work with an unstable unit of measurement, and yet regard their opinions as reliable and scientific! Nothing can be of greater biological importance to a nation (or any other organism) than the way in which it gets its living, that is, its national economy. Some historians regard all history as economics writ large! Our national economy is certainly responsible for widespread poverty in a world of plenty, chronic ill health of the people, universal fear of the future, and recurrent devastating wars. A diagnosis of the underlying faults of an economy producing such results is surely imperative.

Dr. Dane has apparently not noticed that compound interest gives the increase by time to the lender, and "depreciation" the losses by time to the worker or producer. An allowance for the effects of time that penalizes the worker for the benefit of the idler hardly appeals to one's sense of justice or wisdom. A money has been devised by which allowance can be made for the effects of time, recognizing both wastage and increase by time. Wice currency bases the unit of money on the unit of labour and is thus a credit money: it uses a separate symbol for each of the three phases of a unit of money. For example, (1) the pound note (Wice note) is a claim to (2) a pound's worth, while (3) the unused or surplus pound is invested; these three, though interchangeable, are not the same thing. Wice recognizes this and gives the name Basenwice (B) to the wage-note, Standanwice (S) to the money's worth, and Freowice (F) to the invested pound. Complete records can thus be kept of current wages, current supplies, and national reserves; much other information is also obtainable from such records. By combining the Wice currency with the jubilee period (of Mosaic origin) adjustments for short-period movements (of flocks and herds and crops) are made every seven years, and for long-period (human) movements in the fiftieth year. The jubilee period also gives a time-limit to contracts and permits transfer from debt to credit without repudiation. No doubt three symbols could be invented for sterling, but sterling is a debt-commodity money. Wice gives a credit, renewable from week to week and each week, which thus, like the widow's cruse, goes on for ever.

The whole country lives on the primary producer and his product. If the primary producer were in credit he would employ more, pay more, and buy more; unemployment would speedily vanish. While the primary producer gets too little in return for his work, as at present, he cannot keep secondary industry employed. This seems obvious to me, if not to Dr. Dane. Long ago, Adam Smith pointed out that the towns live on the country.

A free medium of exchange (credit money) is more important to the worker and producer than a free land; the latter is dependent on the former. In Genesis we find

a detailed account of how a free people were enslaved, both their lands and their persons, by corn loans (by their own produce from their own land!).

To Dr. O'Day I would point out that Marx was greatly lacking in analytic power, that most of his "capital" is vitiated by two facts: first, that he treated three separate things (machinery, the capitalist or master, and money) as if they were one—capital. He claimed that labour was the source of all values, but accepted the current debt-commodity money. (With a money based on labour the unjust seizure of surplus values by the few is impossible.) In the second place he neglected all kinds of labour other than that productive of commodities (chiefly mass-productive labour); he forgot that goods are produced for use, that use being the sustenance, the education and the development of a nation, and that these are only possible by the work of static or subsistent workers, mainly women and children, and public services, such as police, health, sanitation, education, the churches *et cetera*. Marxian economics is certainly not the last word in scientific economics.

To "M.B., Ch.B." I would say that Douglas Social Credit accepts the present commodity money, with its necessary instability of unit and its injustice in distribution, while it issues credit to the consumers through the retailers; this is a direct encouragement of parasitism and in itself would lead to failure of the plan. If credit is issued, it must be issued to the producer or the worker for work done.

I wish to thank all your correspondents (even the anti-pathetic "Cobbler", who was at least no Laodicean!) for their evidence of interest in the discussion.

Yours, etc.,

Mary C. De Garis.

Geelong, Victoria.
March 18, 1933.

Books Received.

DIAGNOSTIC METHODS USED DURING THE LATER MONTHS OF PREGNANCY AND DURING LABOUR, by J. C. Windeyer, M.D., Ch.M., M.R.C.S., L.R.C.P., F.R.A.C.S., F.C.O.G.; 1933. Sydney: Australasian Medical Publishing Company, Limited. Demy 8vo., pp. 26, with illustrations. Price: 1s. 6d. net.

SEX DETERMINATION, by F. A. E. Crew, M.D., D.Sc., Ph.D.; 1933. London: Methuen and Company, Limited. Foolscap 8vo., pp. 138. Price: 3s. 6d. net.

Diary for the Month.

Apr. 3.—New South Wales Branch, B.M.A.: Organisation and Science Committee.
Apr. 4.—New South Wales Branch, B.M.A.: Council.
Apr. 5.—Victorian Branch, B.M.A.: Branch.
Apr. 5.—Western Australian Branch, B.M.A.: Council.
Apr. 6.—South Australian Branch, B.M.A.: Council.
Apr. 7.—Queensland Branch, B.M.A.: Branch.
Apr. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Apr. 18.—New South Wales Branch, B.M.A.: Ethics Committee.
Apr. 19.—Western Australian Branch, B.M.A.: Branch.
Apr. 20.—New South Wales Branch, B.M.A.: Clinical Meeting.
Apr. 21.—Queensland Branch, B.M.A.: Council.
Apr. 27.—South Australian Branch, B.M.A.: Branch.
Apr. 27.—New South Wales Branch, B.M.A.: Branch.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", page xvi.

Fremantle Hospital, Fremantle, Western Australia: Resident Medical Officers.

Hamilton and District Base Hospital, Victoria: Resident Medical Officer.

Mater Misericordiæ Children's Hospital, Brisbane, Queensland: Resident Medical Officer.

Newcastle Hospital, Newcastle, New South Wales: Junior Resident Medical Officers.

Saint Vincent's Hospital, Melbourne, Victoria: Medical Officers.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
New South Wales: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
Victorian: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
Queensland: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Toowoomba Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
South Australian: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
Western Australian: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
New Zealand (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

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